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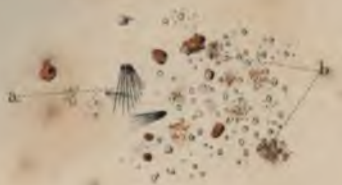

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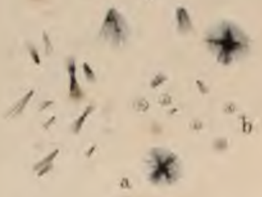

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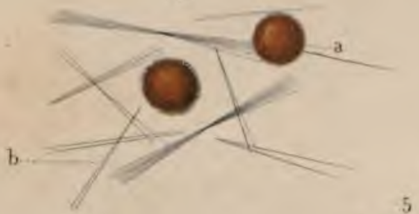

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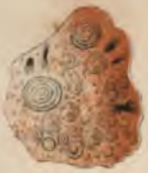

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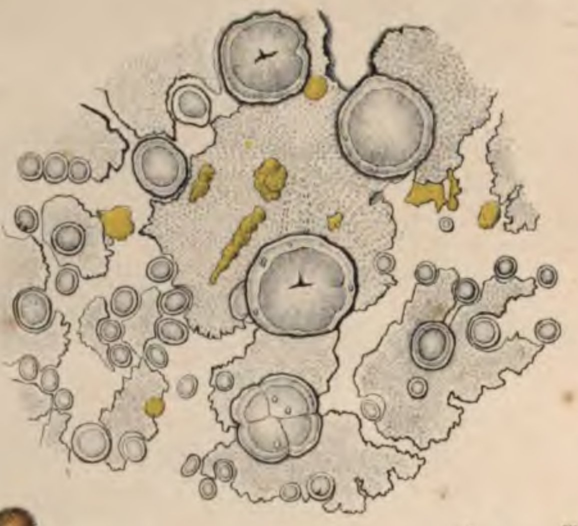

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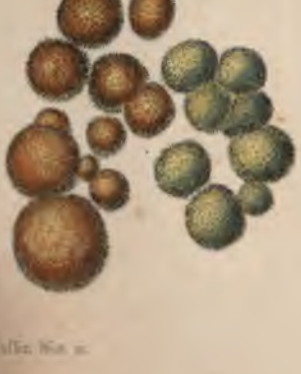

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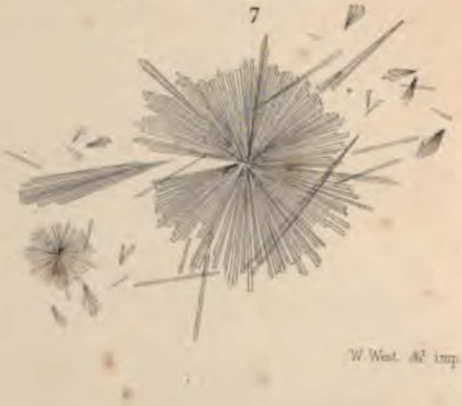

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A

CLINICAL TREATISE

ON

DISEASES OF THE LIVER.

BY

DR. FRIED. THEOD. FRERICHS,

PROFESSOR OF CLINICAL MEDICINE, FORMERLY IN THE UNIVERSITY OF BRESLAU, AND NOW IN THE UNIVERSITY
OF BERLIN; ETC., ETC.

IN TWO VOLUMES.

VOL. I.

TRANSLATED BY

CHARLES MURCHISON, M.D.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON; ASSISTANT PHYSICIAN TO KING'S COLLEGE HOSPITAL,
AND TO THE LONDON FEVER HOSPITAL.

THE NEW SYDENHAM SOCIETY,
LONDON.

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AUTHOR'S PREFACE.

With the present Clinical Treatise on Diseases of the Liver, it is intended to commence a series of Works which shall contain the results of my clinical experience and pathological observations. The manner in which I propose to treat the subject, will appear from the works themselves; and it, therefore, seems needless to discuss this any more in detail at present; it is only necessary for me to make a few brief explanatory remarks.

At the present day, it is agreed, that the science of life is undivided, and that no real defined limits exist between the varying phenomena of health and disease, but that both are governed by the same laws. We endeavour to investigate the conditions under which disease takes place in the same rigidly empirical manner, that we study the general vital processes of which disease is merely a fragmentary constituent. In addition to simple observation at the bedside of the patient, we have recourse to the assistance of physical, chemical, microscopical, and experimental modes of investigation, in order to collect materials for the construction of a scientific system of medicine.

There can be no doubt, that a great advance has been made by this mode of research, which, although not novel, has at no time been adopted to the same extent, and with such ample means at our disposal, or attended by equally important results, as during the last ten years. Our general views of disease have been simplified since we have ceased to disconnect it from the phenomena of life, as something foreign and endowed with a peculiar and individual exist-

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practice. This explains why the subject of icterus is treated at such length; and why I have annexed to this the consideration of acholia and of acute atrophy of the liver, which last might with more propriety have been considered under the head of inflammation of the organ. At the end of the work, I shall classify the various morbid processes according to their mutual relations.

The present volume contains the historical account of diseases of the liver, the definition of the dimensions and weight of the organ, physical diagnosis, the chapters on icterus, acholia, acute and chronic atrophy, the fatty liver, the pigment-liver,* and the hyperæmias and hæmorrhages of the liver.

The more important diseases of the liver will find a place in the second volume:—Inflammation with its consequences, cirrhosis, the colloid or lardaceous degeneration, morbid growths, the diseases of the bile ducts and of the portal veins, &c. At the conclusion of the entire work, I purpose to collect the general results derived from the isolated data, and to point out their value in reference to the pathology and treatment of the organ.

Lastly, I would desire to thank those gentlemen who have aided me in the preparation of this work, and particularly my friends and colleagues, Herr Reichert, councillor of state, and Professor G. Staedeler of Zurich. I am indebted to the former gentleman's extensive acquaintance with the entire subject of minute anatomy for repeated suggestions and assistance, while his skill in making preparations has essentially contributed to the success of the Atlas of Plates drawn by Herr Assmann. My friend Staedeler has assisted me on many occasions with chemical advice, and I have to thank him for the elementary analyses of the abnormal products of metamorphosis detected in the liver and in the urine.

Dr. Valentin has performed a large proportion of the chemical

---

* Under the head of the pigment-liver, I have been compelled to expatiate and to describe derangements, which may be said to have no direct connection with hepatic disease. I trust that the rare opportunities which I have enjoyed of observing large accumulations of pigment in the blood, and the small number of cases which as yet have been accurately described, will be regarded as an excuse for this digression.

A

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repeatedly confirmed, it has been concluded, that there is an intimate relation between the biliary acids and the bile-pigment, and that in fact the former become converted into the latter when subjected to the influence of certain agencies; and it has been thought, that, under certain pathological conditions, the biliary acids normally present in the blood are transformed into bile-pigment. The detailed account of Frerich's experiments will be found in the section which treats of the Theory of Jaundice, and in the Appendix to the present volume.

Since the appearance of Frerich's work, these views as well as the facts upon which they are founded, have been called in question by Dr. Kühne, (*Virchow's Archiv., 3rd and 4th Part of Vol. XIV., Sept.* 1858); and as an abstract of Kühne's paper has lately appeard in an English Medical Journal, and may have some influence with English readers (*Archives of Medicine, edited by Dr. Beale, Vol. I., p.* 342, *March,* 1859), it demands a brief notice here. Kühne maintains that biliary acids do constitute an integral part of jaundiced urine, and he attributes the circumstance of their not having been hitherto demonstrated, to the insufficiency of the tests employed for the purpose. The method which he has followed has been that of Dr. Felix Hoppe, an account of which will be found in the thirteenth volume of "Virchow's Archives," and in the first volume of the "Archives of Medicine," edited by Dr. Beale (p. 344). The following conclusions are arrived at by Kühne as the result of his investigations.

1. In jaundice resulting from closure of the *ductus communis choledochus*, the urine *always* contains biliary acid as well as bile-pigment.

2. The presence of biliary acids is a peculiarity of jaundiced urine only. They are never present in normal urine.

3. The biliary acids are not decomposed in the blood. In whatever manner they find their way into this fluid, they are afterwards excreted unchanged by the kidneys. Glyco-cholate of soda injected into the veins passes out in the urine unchanged. On the other

hand, according to Frerichs, when glyco-cholate of soda or any other pure bile-acid salt is thrown into the veins, bile-pigment appears in the urine, while unchanged biliary acids can never be detected.

4. After the injection into the veins of colourless solutions of the biliary acids or of their salts, bile-pigment may appear in the urine, as stated by Frerichs; but the colouring-matter which is developed under such circumstances is due to the property possessed by the biliary acids of dissolving the blood-corpuscles and of thus setting free a quantity of hæmatine, which, being acted on in some unexplained way by the biliary acids or their salts, is converted into bile-pigment. In support of this view, Kühne appeals to the circumstance of albuminuria or hæmaturia having been usually observed after the injection of biliary acids into the blood, and to the following experiment. A solution of hæmato-globuline containing a minute trace of glyco-cholate of soda was injected into the veins of a dog, and it was found that the urine subsequently secreted gave a splendid reaction of bile-pigment with nitric acid. Kühne even thinks it highly probable, that a considerable number of blood corpuscles is, under ordinary circumstances, destroyed in the liver, and that the colouring-matter of bile is derived from the hæmatine set free by the solution of these corpuscles.

5. The bile acids all pass off with the fæces, and are not reabsorbed from the intestine.

Quite apart from the correctness of Frerichs's theory of icterus, which, by the way, is only advanced as one that is highly probable, it is obvious that we have here to do with a question of facts, and that Kühne's facts are diametrically opposed to those brought forward by Frerichs. It is due, however, to Frerichs to state, that the results arrived at by him have been confirmed by several subsequent observers. Dr. Folwarczny, of Vienna *(Zeitschrift der kaiserl. königl. Gesellschaft der Aerzte zu Wien.* 1859. No. 15, p. 225), examined the urine in three cases of jaundice in Prof. Oppolzer's

Clinique, but in all he failed to detect any trace of the biliary acids, although the examination was performed repeatedly, and Hoppe's process adopted in each case.

Professor Staedeler of Zurich, and Dr. Neukomm, have likewise arrived at results similar to those of Frerichs, and have in Frerichs's opinion, completely refuted the statements made by Kühne. The results of their researches are about to be published (*Archiv. für Anatomie, Phys. und Wissenschaftliche Med. von Dr. C. B. Reichert und Dr. E. Du Bois Reymond.* 1860. *Ueber die Nachweisung der Gallen Säuren und die Umwandlung derselben in der Blutbahn. Von Dr. F. Neukomm*); and I am indebted to Professor Frerichs for a manuscript copy of the Memoir in which they are embodied. The first portion of this Memoir is devoted to a comparison of the delicacy of the several tests for the biliary acids, Pettenkofer's, Hoppe's, and that of another recommended by the author, which is a modification of the ordinary process by precipitation with acetate of lead. It is shown that the last of these processes is much more delicate than that of Hoppe's. These several processes are then applied to the examination of two specimens of jaundiced urine from the human subject, and to that of the urine of seven dogs, into whose blood solutions of the biliary acids had previously been injected. In both the specimens of jaundiced urine, Pettenkofer's test gave a negative result, but the author's process indicated the presence of minute traces of the biliary acids. It is shown, however, that the quantity of these acids was so small (in one instance, only 5 milligrammes in 1200 cubic centimètres of urine, or less than $\frac{1}{13}$ of a grain troy in 42 fluid ounces) as to bear a very small proportion to the amount which must have entered the blood. Of the seven dogs into whose blood *large quantities* of the colourless solutions of the biliary acids were injected, in all the urine became coloured with bile-pigment within a certain number of hours after the operation; in none could any bitter taste be distinguished in the urine; in none did the urine exhibit the slightest reaction with Pettenkofer's test; and in two cases only did the author's test indicate the presence of traces of the biliary acids in this secretion. It is contended that these cases confirm the statements made by Frerichs to the effect that the biliary acids are converted in the blood into bile-pigment.

There would appear to have been some important difference between Kühne's experiments and those of Frerichs. Kühne tells us, that in his experiments, the dogs died suddenly soon after the injection into the veins of the colourless biliary acids; whereas it is distinctly stated by Frerichs that the injection of these substances produced little or no change in the deportment of the animals, and that in one case the experiment was repeated four times without entailing any lasting injury. Moreover, the co-existence of a small quantity of biliary acid along with bile-pigment in jaundiced urine, when it does occur, is not opposed to the correctness of Frerichs's theory, for it may easily be supposed that a portion of the biliary acid may remain unconverted into bile-pigment. As to Kühne's opinion, that the colouring-matter which appears in the urine, after the injection into the veins of the colourless biliary acids, is derived from the hæmatine of the blood, it may be observed that, although it is possible that the colouring-matter of the blood may become transformed into bile-pigment, positive proofs are still wanting to show, that such a transformation really takes place. No one has yet succeeded in obtaining bile-pigment from the colouring-matter of the blood. At all events, Kühne's experiments fail in proving that the colouring-matter in the urine originates from this source, and not from a transformation of the biliary acids; and they likewise fail in accounting for the disappearance of the biliary acids injected into the blood, in any other manner than that suggested by Frerichs.

Further observations and experiments on the whole subject are still required; but in the meantime it should be understood, that the main facts adduced by Frerichs in support of his theory of Icterus have received confirmation at the hands of most subsequent observers.

Since the publication of the German edition of the first volume, certain experiments have been performed in Frerichs's laboratory by his assistant, Dr. Valentin, which tend to show, that one of the colouring-matters of bile consists of hæmatine, the substance which is known to be derived from blood-pigment. Valentin has succeeded in detecting crystals of hæmatine in gall-stones, in the bile of men and animals, and in the tissues and secretions of jaundiced patients. The addition of chloroform is found to dissolve the hæmatine with a yellow colour, and from this solution red and brownish-red, lancet-shaped, and rhomboidal prismatic crystals separate, which correspond in every respect with those of hæmatine (*Günsburg's Zeitschrift, Dec.* 1858). From these experiments, Frerichs admits there is an intimate relation between bile-pigment and the colouring-matter of the blood, and even thinks it probable, that the former substance may be developed from the latter. Still he urges, that no one has succeeded in obtaining bile-pigment from the red matter of the blood, and that Valentin's results are not at all opposed to his theory of the convertibility of the colourless biliary acids into bile-pigment.

It may be mentioned, that although only the first German edition has as yet been published, most of the corrections and additions for the second edition, have, through the kind co-operation of the author, been incorporated in the present English translation. Unfortunately, the alterations for the section on the "Theory of Jaundice," which have been suggested by the statements of Kühne, and by the recent observations of Staedeler, Neukomm and Valentin, were not received until long after that portion of the translation had passed through the press. A list of these alterations will be found immediately after the Table of Contents.

The German edition of the work is illustrated by forty-two woodcuts and by an Atlas of twelve coloured plates. Twenty-nine of the more important woodcuts have been reproduced by Dr. Westmacott, who has succeeded in improving the style of their execution, without at all interfering with the objects they are designed to represent. The republication of the Atlas would have involved too great an outlay, but Messrs. Williams and Norgate of 16, Henrietta Street, Covent Garden, will supply copies of it to any members of the New Sydenham Society. A few figures representing the crystalline forms presented by leucine and tyrosine have been copied from the

Atlas, and form the frontispiece to this translation. Three other figures (Figs. 30, 31, and 32) have been copied from the same source, and added to the list of woodcuts.

It has been the object of the translator to adhere as closely as possible to the original text, so far as this was compatible with readable English. Throughout the work, the measurements and weights adopted by the author have been retained; but their equivalents in English measurements and weights have everywhere been added within brackets. A similar plan has been followed in cases where the size of objects is compared to that of Prussian coins. Notes have been appended to the various tables of weights and measurements, showing how these may readily be reduced to their corresponding English equivalents. A few other short notes have been added, giving an account of the composition and properties of the German Spas, and of various preparations of the Prussian Pharmacopœia, and also explaining several matters referred to in the work, with which few English readers can be expected to be familiar. It has been thought that, by these notes of explanation, the practical utility of the work might be enhanced.

79, Wimpole Street, W.,
June, 1860.

EXPLANATION OF FRONTISPIECE.

Fig. 1. Disintegrating glandular cells of liver (*b*) and bundles of needle-shaped crystals of tyrosine (*a*), from a case of acute atrophy of the liver. See Observation XIV., page 202.

Fig. 2. Blood from the hepatic vein of the same case as Fig. 1: *a*, crystals of tyrosine adhering in radiating bundles; *b*, normal blood-corpuscles.

Fig. 3. Crystals of tyrosine which separated from a decoction of the liver upon cooling; from the same case as Figs. 1 and 2: *a*, crystalline masses impregnated with colouring-matter; *b*, long needle-shaped crystals.

Fig. 4. A fragment of the parenchyma of the liver, from a case of acholia arising from impermeability of the bile ducts. The secreting cells have become almost entirely disintegrated, and their place is supplied by bundles of acicular crystals of tyrosine, and globular masses of leucine deposited in concentric layers. See Observation XIX., p. 240.

Fig. 5. A drop of urine passed by a patient labouring under acute atrophy of the liver, and evaporated upon an object-glass. It exhibits leucine deposited partly in globular masses, with concentrically-thickened walls and fissured surfaces, and partly, in finely-striated laminæ, and also greenish-yellow crystals of tyrosine. See Observation XV., p. 204.

Fig. 6. Greenish-yellow globular masses of crystals of tyrosine deposited spontaneously from the urine of the same case as Fig. 5, after standing in the cold for twenty-four hours.

Fig. 7. Crystals of pure tyrosine obtained by re-crystallization from the same sediment as mentioned in the description of Fig. 6.

N.B.—A List of the Woodcuts is appended to the end of the volume, page 401.

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ALTERATIONS FOR SECOND EDITION.

PAGE 85, lines 18 and 19; page 89, lines 18 and 19, and page 93, lines 11 and 12; substitute, "Through some alteration in the metamorphosis of substances contained in the blood."

Page 89, For paragraph commencing line 20, substitute:—

"The bile-pigment is so intimately related on the one hand to the red "matter of the blood, and, on the other, to the colourless biliary acids, as "to justify us in referring its origin to one or the other of these sources.

"The intimate relation subsisting between the bile-pigment and the "colouring-matter of the blood is indicated by facts which have been "already mentioned, but more particularly by observations which have "been recently made in my laboratory by Dr. Valentin (*Günsburg's* "*Zeitschrift, Dec.* 1858), according to whom a portion of the colouring-"matter of the bile dissolves in chloroform, and from this solution a "crystalline substance may be obtained presenting all the characters of "hæmatoidine. From this it appears possible, nay probable, that, as in "extravasations, hœmatoidine may be developed from blood-pigment, so "in like manner, in the vascular system and in the liver, the colouring-"matter of bile may originate from the same source. Hitherto, how-"ever, no one has succeeded in obtaining bile-pigment directly from "the red matter of the blood."

Page 89, at commencement of last paragraph, for "This view," substitute "The second view."

Page 91, to end of note which commences on page 90, add, "The state-"ments which have been made by Dr. Kühne (*Virchow's Archiv*, xiv., p. "310) in opposition to the correctness of this view, have been completely "refuted by Dr Neukomm (*Archiv für Anatomie und Physiolpgie*. Leipzig, "1820)."

Page 96, at end of note which commences on preceding page, add, "The "assertions of Kühne and Hoppe to the effect that the biliary acids pass "unchanged from the blood into the urine, have been completely refuted "by Neukomm and Staedeler."

A
CLINICAL TREATISE
ON
DISEASES OF THE LIVER.

CHAPTER I.

HISTORICAL INTRODUCTION.

It is very interesting to trace historically the views which medical men at different periods have formed concerning the functions of individual organs of the body, and the diseases to which these organs are liable. The tree of knowledge, with its flowers and fruit, appears less strange to us, when we follow its roots, as they branch out, at one time more deeply, and at other times less so, into the region of history, and when we search out the springs which have made it bear fruit. That which at the present day is brought forward as new has not unfrequently been known centuries ago.

With regard to no organ does history attest a change of views in a more remarkable manner than in the case of the liver.[*] This organ and the portal venous system attracted at a very early period the attention of physicians. More from vague conjectures than upon clearly established grounds, the seat of various functions, of great importance both in health and in disease, was transferred to this powerful glandular organ, and to the extensive system of vessels, which is intimately connected with the gastro-intestinal canal. By the ancients the liver was regarded as the central organ of vegetative life.[†] Galen looked upon it as the focus of animal heat, and as the organ intended for the formation of blood, and for the origin of the veins.* According to him, the metamorphosis of chyle into blood commenced in the portal veins, but was completed in the liver, which organ, during the process, separated as waste matter from the blood the yellow and the black bile, the former passing to the gall-bladder, the latter to the spleen.†

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[*] See Beau, Arch. génér. de Méd. 1851.

[†] Plato, in his Timæus, calls the liver a *θρέμμα ἄγριον*, on account of

Galen's views underwent scarcely any modification by the Arabian physicians, and remained undisputed until the middle of the seventeenth century. Even Vesalius, who, by his anatomical researches, contributed more than any one else to the overthrow of the doctrines of Galen, did not venture to oppose his physiological theories respecting the liver; he only denied the assimilative property attributed to the portal veins.‡

The attempts of Argentieri § to restrict the functional importance of the organ met with no support.

The discovery of the lacteal vessels by Aselli in 1622, and of the thoracic duct by Prequet in 1647, first imparted a severe shock to the views of Galen. A way had been discovered, by which the chyle was conveyed from the bowel into the blood quite independently of the portal vein and of the liver; henceforth, neither of these structures appeared to serve any purpose in sanguification. Bartholin || and Glisson ¶ were the first who enunciated in a decided manner this opinion. It rapidly spread; and the more readily, inasmuch as the revolutions, just effected in the views of physiolo-

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its importance in vegetative life, and in opposition to its asserted spiritual signification. See Galen, de dogm. Hippocr. et Platonis.

* De usu partium, libr. IV. In hepate, quod supponitur venarum principium esse, primum sanguificationis instrumentum.

† Prius elaboratum in ventriculo alimentum venæ ipsæ deferunt ad aliquem concoctionis locum communem totius corporis, quem hepar nominamus. De usu partium, libr. IV.

‡ De corporis humani fabricâ, 1542, libr. III. 267, libr. V. 508. Quod vero iidem rami, priusquam jecori succulentum id porrigant, rudem aliquam sanguinis formam cremori seu succulento illi conferant, et ut Galenus attestatur, modo jecori simillimo istud præparent, non facile concessero.

§ De erroribus veter. med., Flor. 1553; and, Comment. tres in art. med., Galeni. Paris, 1553.

|| Vasa lymphatica nuper in animantibus inventa et hepatis exequiæ. Paris, 1653.

¶ Anat. hepat., p. 289. Edit nova. Amstelodami, 1665.

gists by the labours of Harvey,* bestowed an importance hitherto unknown upon the organs of the chest.

Riolan, indeed, and after him De Bils, endeavoured to defend the important part played by the liver in sanguification, in opposition to the views of Bartholin; but Bartholin came victoriously out of the contest, and wrote for the liver a humorous epitaph, in which the end of its dominion was announced, and in which its function was declared to be henceforth limited to the secretion of bile.†

For almost two centuries this opinion continued generally to prevail. Swammerdamm, it is true, endeavoured to advocate once more the ancient theory, yet with so little success, that Boerhaave observed: "Dudum in meliori parte Europæ obsolevit hæc sanguificatio nunquam ab eo viscere expectanda."

It was reserved for the experimental physiology of the present century to extend the boundaries of our knowledge in this matter, and to reproduce, in a more novel and exact form, opinions which, for a long period, had appeared buried in oblivion. The first step in this direction was made by Magendie‡ and Tiedemann,§ who furnished the proof that the absorption of nutritive matters is not solely the work of the lacteal vessels, but that a part of the food digested in the gastro-intestinal canal is taken into the blood through the portal vein. Tiedemann and Gmelin moreover, by a series of careful experiments, arrived at the result, that "the liver must also be regarded as the organ for the assimilation of substances which have been absorbed from the intestine."

The more recent investigators who have paid attention to this subject, such as Blondlot,|| Claude Bernard,¶ Lehmann,** C. Schmidt, and others, have been obliged to corroborate the opinion as to the

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* Harvey himself attached little weight to the lacteals, and upheld the views of Galen in respect to the liver.

† Defensio lacteorum et lymphaticorum contra Riolanum. Hafniæ, 1655. Bartholini responsio de experimentis Bilsianis et difficili hepatis resurrectione. Hafniæ, 1661.

‡ Précis élémentaire de physiol. Tom. II., p. 268.

§ Tiedemann et Gmelin, Versuche über die Wege auf welchen Substanzen aus dem Magen-und Darmcanal ins Blut gelangen. Heidelberg, 1820.

|| Essai sur les fonctions du foie. Paris, 1846.

¶ Leçons de physiologie expérimentale appliquée à la médecine. Paris, 1855.

** Physiolog. Chemie., Bd. III.

participation of the portal vein in the absorption of materials from the intestine into the system, although much remains doubtful as regards the extent and importance of this participation. It is to be regarded as nearly proved that water, salts, sugar, albuminous and colouring-matters are, for the most part, conveyed to the blood by venous absorption; and also that the greatest part of the fat reaches the blood through the lacteals. But it is still a matter of question, in what way the most important principles of nutrition, the albuminous, are absorbed. Claude Bernard and the majority of the French investigators make their absorption take place through the veins, whilst Lehmann, Schmidt, and Ludwig are convinced that it is through the lacteals.

Another point which has not been sufficiently investigated, is the influence which the hepatic parenchyma exercises over the substances which pass through it. According to the experiments of Claude Bernard, Mialhe, and others, the carbo-hydrates, no less than the albuminous principles, in their passage through the portal system, undergo important changes from the action of the liver, by means of which they first become fitted for the formation of blood. A detailed proof of this metamorphosis has not, however, as yet been given with sufficient clearness.

At the conclusion of the work, we shall return to this and to other physiological questions of a general nature, and shall bring together such materials for their solution as present themselves; in the meantime we shall only make a few historical remarks, showing how the important part played by the liver in the absorption and elaboration of the products of digestion came by degrees to be again recognised.

Whilst experiment proves that the liver exercises an indirect influence over the formation of the blood, there are not wanting observations which seem to indicate that it participates directly in the generation of the elementary principles of this fluid, although, taking everything into consideration, these observations cannot be regarded as decisive. Among these may be mentioned the observations which Reichert,* E. H. Weber,† and Kölliker‡ have made upon embryos, and upon frogs wakened up out of their winter's sleep, in reference to the development of blood-corpuscles in the liver. These observers found in this way a remarkable confirmation of the differences between the blood of the portal and hepatic veins, which had been authenticated by the carefully repeated chemical analyses of Lehmann,* and by the changes which, according to the observations of Moleschott, the entire blood undergoes after the extirpation of the liver.

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* Entwickelungsleben in Wirbelthierreiche. S. 22.

† Berichte der königlich sächsischen Gesellschaft der Wissenschaften Leipzig, 1850. S. 15, bis. 20.

‡ Henle's und Pfeufers Zeitschrift. Bd. IV., S. 147, ff.

Besides this direct and indirect participation in the formation of blood, we have very recently become acquainted with the part played by the liver in certain metamorphic processes of an intermediate nature, which are of the greatest importance, as furnishing a deeper insight into health and disease. Bernard† has shown, by a series of accurate and precise experiments, that, notwithstanding that an animal is fed upon nitrogenous food solely, there is always formed in the liver, in addition to bile, a considerable quantity of sugar, which passes into the mass of the blood, to serve an ulterior object, and which appears as necessary for the healthy performance of vital processes as are other metamorphoses, the products of which are thrown off from the system by secreting organs.

In addition to this elimination of sugar from the complex atoms of the albuminous principles, other chemical changes take place in the liver, of which we will hereafter learn the importance. This is shown by the existence in the gland of inosite, hypoxanthine, and uric acid, and further by observing in it in the course of many diseases, the occurrence of cystine, and large quantities of leucine and tyrosine, and lastly, by the remarkable abnormalities in the composition of the urine which are observed in certain diseases of the liver. Besides all this, the important part which the liver plays in the production of animal heat has found a new defender in Claude Bernard.‡

The liver has consequently again ceased to be regarded as merely an organ for secreting bile. The views of Galen, which Bartholin believed he had overthrown for ever, although modified and circumscribed, have risen into new life and significance. There can now be no doubt, that in this gland processes go on, which exercise an important influence over the principal vegetative functions—

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* Berichte der königlich sächsischen Gesellschaft der Wissenschaften 1851. S. 131, ff.

† Nouvelle fonction du foie, &c. Paris, 1853.

‡ Leçons de physiol., Expérim. I., p. 199.

sanguification and the metamorphosis of tissues; what remains to be done is to ascertain at the sick bedside, and by means of experiment, the extent of these processes, and to fix with precision their influence upon health and disease.

As might have been expected, this change of opinions concerning the physiological relations of the liver could not fail to react upon the views entertained as to its pathology. In these views similar changes are met with; only they are less remarkable than in the case of the physiological opinions, because clinical observation must always be directed to the secretion of bile as the chief function of the liver.

In the pathology of the ancients, and particularly of Galen, the liver and the portal system served as the starting-point of manifold disturbances. There were described not only a host of anatomical and functional lesions of the organ itself, such as inflammation, abscess, obstruction of the ducts, and the different conditions resulting from intemperance, but a large proportion of constitutional diseases were referred to the same source. Abnormal conditions of the liver were regarded as the principal cause of changes in the constitution of the blood. "*Sanguificatio vitiatur hepate vitiato.*" Plethora, anæmia, cachexia, and dropsy, were attributed to certain changes in the activity of this gland. A further cause of general diseases was found in the products of the secretion of the organ, the yellow and the black bile, which under a humoral pathology, had a mighty importance as elementary constituents of the organism. The yellow bile, it was thought, would induce acute diseases, running a rapid course and accompanied by a high temperature, such as erysipelas, &c.; while, on the other hand, black bile was believed to give rise to chronic diseases, such as mental derangement, apoplexy, convulsions, &c. No wonder that in this way, the liver came to be regarded as the centre of a large proportion of pathological processes. Throughout the pathological works which appeared from the time of Galen down to the middle of the seventeenth century, this organ was looked upon as the seat of the mind itself. No one dared to question the grounds of this dogma, although in a few instances it was extended or modified. Even in the year 1626, Riolan called upon physicians diligently to study the liver, as the *vitæ et nutricatûs fundamentum.*

The discovery of the lacteals, effected a change in physiological views, which was necessarily followed by a reaction in the theories of disease. One of the foundation-stones upon which the artificial fabric of Galenic pathology had rested, was removed; opinions, which through a lengthened period had apparently acquired the value of facts, became untenable; and novel points of inquiry were opened up. Although the functional importance of the liver had necessarily become circumscribed, other channels of absorption before unknown had been discovered. Simultaneously with this, the brilliant work of Harvey upon the circulation of the blood was attracting much attention, and promised an explanation of many phenomena.

It was Bartholin,* the strenuous opponent of the Galenic theories, who had the merit of undertaking a revision of the science of medicine under the light of the new discoveries in physiology. With a degree of caution worthy of all praise, he declared that the ancient medical doctrines were not overthrown, but only elucidated; that the causes of disease were better understood and more correct than formerly, and therefore more easily obviated; and that especial attention was to be given to the heart as the organ for the circulation of the blood. It was, however, admitted that abnormal conditions of the blood might result from obstruction and other diseases of the liver, although this organ did not take any part in sanguification. The obstinate adherents of Galenic medicine in vain endeavoured to combat these innovations; the views of physicians became modified more and more, and as a substitute for the dogmas formerly prevalent, they hastened to turn the discoveries of the day to a theoretical and practical account.

There is a tendency in the human mind to overrate the importance of new discoveries, and in this way to make an improper use of them. Hence, we can quite understand how crude medical theories should have succeeded the revolutions above-mentioned, in the departments of anatomy and physiology, and the more so, as, at the same time, a more correct knowledge of natural philosophy and chemistry was beginning to shed a new but deceitful light upon many questions. For practical medicine in general, and for the study of diseases of the liver in particular, there commenced an unproductive period, which, trusting to theory more than to actual observation, always failed in obtaining a firm foundation in facts. The followers of Sylvius, the Iatrochemists, as well as the Iatrophysicists, left behind them no fruits of direct investigation on the subject with which we are now engaged. The theory of Franz de le Boë Sylvius, who maintained that the fermentation of the juices secreted by the spleen, pancreas, and liver, was of great importance for chylification and sanguification, both in health and in disease, acquired a wide-spread influence, only because it compensated for the opposition bestowed, since the discovery of the thoracic-duct, upon the Galenic method of explaining many diseases, which, it was believed, must necessarily result from a disturbance in the functions of the liver. Not much more resulted from the writings of Sydenham, except that he re-established the Hippocratic method of clinical observation, and removed the abuses which had resulted from crude sources of information. Considering his great distinction in the practice of medicine generally, he bestowed but little attention upon the diseases of the liver.

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* An hepatis funus immutet medendi methodum. Hafniæ, 1653.

Meanwhile, and partly even before this period, a better future was gradually being opened up through the anatomical and pathological investigations to which physicians had begun to devote themselves with increasing zeal. In this way there was accumulated by degrees materials, from which a future age was able to construct the foundation of a pathology of liver-diseases founded upon facts, although, certainly, one-sided. Even before the general revolution in medical opinions, Benivieni,* Vesalius,† and Fallopius‡ had, from their anatomical studies, collected contributions which served to cast a new light over several forms of hepatic disease. They were the first to give accurate descriptions of gall-stones, and of the consequences which resulted from their detention in the gall-bladder. Vesalius reported a case of bursting of the portal vein in consequence of cirrhotic degeneration of the liver; he noted the prejudicial effects of spirituous liquors upon this organ, and the connection between intumescence of the spleen and disease of the liver.

The writings of Glisson § contained some valuable observations upon the subject of tumours of the liver in rachitis; abscesses and concretions were treated of by Bartholin; || malignant jaundice by Baillou,¶ a very able physician, and especially by Th. Bonnet, in his large and comprehensive work.** The last of these authors re-

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* De abditis morborum causis. Cap. 3, pp. 94, 140, 263.

† Epistola de radie. Chin. Basil., 1546, p. 642.

‡ Observ. Anatom., p. 401.

§ Anatom. hepat.

|| Historia anat., Cent. VI.

¶ Ballonii opera. om. Genev., 1662, Tom. I., p. 188.

** Sepulchretum anat. Genevæ, 1679.

corded an instructive series of cases of jaundice depending upon obstruction, and divided his observations under the heads of Inflammations, Tumours, Scirrhus, Obstructions, Cysts, Calculi, &c., of the Liver. It is true that these observations, in many places, require sharp criticism; yet, as the first rudiments of our knowledge, they are of much interest. Bonnet's description of Cirrhosis, indeed (*Sect. I., Observ.* 4), leaves little more to be desired. Sylvius, and afterwards Bonnet, discussed at full-length the importance of the liver in general pathology.*

Although, in the present state of our knowledge, these early dawnings of anatomico-pathological investigation may appear incomplete and defective, yet they were of great weight at a time when every well-grounded fact removed entire series of false theories, and formed starting-points for new studies rich in results.

All the information concerning diseases of the liver, which, in the course of time had been obtained through these investigations, J. B. Bianchi endeavoured to bring together in his work entitled, *Historia hepatica seu Theoria et Praxis omnium morborum hepatis et bilis.* This work, although it went through three editions, contained much that was crude and carelessly done, and therefore deserved the severe criticism which it suffered at the hands of Morgagni and Haller. Its influence upon the further study of hepatic diseases was limited, and the more so, as, soon after its appearance, transcendent geniuses brought facts to light which necessarily eclipsed all that had gone before. H. Boerhaave and G. E. Stahl appeared in the field of clinical medicine; J. B. Morgagni in that of pathological anatomy.

H. Boerhaave, who, in combining the artificial system of Sydenham with the fundamental investigation of nature, was a bright example to later times, paid particular attention to the diseases of the liver, because in them he believed he had discovered the source of a large proportion of chronic diseases.† A disturbance of digestion, resulting from a deficient secretion of bile, Boerhaave believed to be the cause of a faulty chylification, from which proceeded dropsy, cachexia, leucophlegmasia, &c.* In addition to defective assimilation, the stagnation of blood in the portal vein was considered as a second source of morbid conditions of the liver. The retardation of the blood at this part of the vascular system was, in Boerhaave's opinion, independent of the heart's action,† and was brought about by the contractility of Glisson's capsule, and the pressure of the abdominal viscera. Hence, it was thought, stagnations of blood often occurred at this place, and black bile was formed, which gave rise to obstruction of the viscera, especially of the liver, to hypochondriasis, melancholia, and many other diseases.‡ In his treatise *Hepatitis et Icterus multiplex*, which Boerhaave declared to be one of his best works, there is a comprehensive account of the special pathology of the liver. It is remarkable, however, that the changes in physiological views among ancient physicians should have failed to effect any essential alteration upon pathology.

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* Wenn die Leber kalt sei und die sauren Säfte, die von der Milz herkämen, nicht verarbeite, so entstehe eine mangelhafte Fermentation, der Chylus werde unvollkommen, das Blut bleibe serös und Wassersucht bilde sich. Wenn die Leber heiss sei, so entstehe unpassende Fermentation, welche Fieber, Entzündung Putrescenz bedinge, unter Umständen Icterus, Diarrhöe, Cholera, Dysenterie einleite. Bei verstopfter und scirrhotischer Leber entständen Cruditäten verschiedener Art (p. 26, *et seq).*

† Prælect. academ. Ed. Haller, Vol. III., p. 186. Duo viscera sunt, a

Almost at the same time, G. E. Stahl,§ relying less upon facts than upon theoretical reasoning, endeavoured to claim for the liver a very comprehensive pathological importance. He called in question the opinion as to the exclusive participation of the lacteals in the absorption of nutriment, which, since the discovery of the thoracic duct, had gradually become generally admitted; and, at the same time, he maintained that as large a quantity of chyle was carried with the blood through the mesenteric veins to the liver, as that which found its entrance into the system through the lacteals. The absorption of improper ingesta into the vena porta was regarded by him as an important source of the changes in the contents of this vessel. Neither Stahl nor Boerhaave believed that the circula-

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quibus fere anne omne morborum chronicorum genus oritur, pulmo a quo tabes, hepar, a quo innumerabiles lenti morbi. *Ibid.* p. 190. Atqui ex centum morbis chronicis vix unus, cujus princeps sedes non sit in hepate.

* Quanquam bilis languet, nata est origo morbi alicujus chronici; chylus enim non potest legitime præparari, inde hydrops, cachexia, leucophlegmasia, &c.

† Sanguis enim venæ Portarum . . . . amittit omnem a corde acceptum impetum, *loc. cit.* III., 183. Cum sinus Portarum pariter sit cor hepatis uti cor dictum universo corpori. *Ibid.* p. 115.

‡ Prax. medic. pars V., p. 48 *et seq.*

§ De vena portæ, porta malorum hypochondriaco—splenetico—suffocativo, hysterico—colico hæmorrhoidariorum. Hal., 1698.

tion of the blood in the portal vein depended upon the heart's influence, but upon that of the respiratory organs, and upon a peculiar tonic motive power, which was imputed to the abdominal organs, and especially to the spleen, the intestines, and the mesentery, as well as to the coats of the vessels themselves.

The diseases of the vena porta—to which Stahl referred those of all the abdominal organs with which this vessel was connected—were reduced to four elementary forms, from which was constructed a theory of the most heterogeneous pathological processes:—1. Abnormal capacity; contraction and dilatation. 2. Increased consistence of the blood, resulting from the absorption of ingesta which are acid, feculent and thick with mucus, from sluggish respiration, &c. 3. Passive stagnation. 4. Active stoppages in the circulation of the blood, resulting from the above-mentioned relaxation and constriction, and from anomalous changes in position of the thickened blood.

Although the physiological proof of these views, and of the consequences resulting from them, necessarily remained defective, still the doctrines involved in them were not without some influence upon the succeeding age. Stahl, it is true, found but few supporters among his contemporaries, or even afterwards among medical men generally; but the theory of abdominal plethora, and of the stagnation of blood in the bowels, which at a later period was elevated by Kämpf* into the doctrine of Infarctus, remained a standing and important article of pathology, which even at the present day attracts many supporters among physicians, and among the lay public has acquired the power of a confession of faith. The careful study of abdominal diseases was greatly prejudiced through this vague doctrine offering an easy explanation for every case.

Whilst, on the part of physicians, many things were being done which must have retarded rather than have advanced progress, J. B. Morgagni† was collecting a rich store of anatomical facts and clinical observations, for which he secured an imperishable value, even in recent times, owing to their clearness and to the care with which they were worked out. For most of the diseases of the liver, we find in his works, the outlines of what is currently accepted at the present day.* After the time of Morgagni, anatomical investigation was pursued with greater zeal, and acquired increasing importance in the clinical study of diseases generally, and of those of the liver in particular. The several structural changes of this gland became more clearly understood, and distinguished from one another; and their nature and consequences began to be placed upon a surer foundation, for which end the increasing study of the finer tissues by means of the microscope furnished still better and more convenient ways and means. The information upon this subject, which was contained in the works of Lieutaud, A. Portal, Mathew Baillie, Carswell, Andral, Cruveilhier, Rokitansky, and many others, formed a solid nucleus, around which the more modern pathology of the liver has arranged itself.

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* Abhandlung von enier neuen Methode, die hartnäckigsten Krankheiten, die ihren Sitz im Unterleibe haben, besonders die Hypochondrie, sicher und gründlich zu heilen. Frankfurt und Leipzig, 1787.

† De sedibus et causis morborum per anatomiam indagatis. Ebroduni, 1779.

At the very time that the anatomical element was acquiring weight and influence in the investigation of liver-diseases, the pathological importance of the organ in diseases generally was gradually losing ground. It is true, that Van Swieten and his contemporaries upheld the views of Boerhaave in their essentiality; it is true, likewise, that at a later period, owing to the then prevailing epidemic constitution, the bile assumed a degree of importance in pathology such as it never had before; and to such an extent was this importance exaggerated, that even Kämpf's doctrine of Infarctus found a hearing. Still all this was of short duration; the labours of anatomists, the chemical analyses of the bile, the physiological experiments upon the secretion and uses of this fluid, and the study of the digestive process, year by year, removed the grounds, on which the comprehensive general importance of the liver, both in health and in disease, had hitherto been based.

After the discovery of the elementary composition of the bile, the liver came to be regarded as the organ which purified the blood of its carbonaceous products, and as being in this way vicarious with the lungs; scarcely a trace remained of the views entertained by the old physicians, that the organ exercised a manifold influence over the formation of the blood. The study of liver-diseases acquired in this way the simple anatomical character which we recognise at the present day: and owing to this study being cultivated more on the dissecting table than at the sick bedside, the investigation of the more obscure functional disturbances of the liver itself, and of its participation in other acute and chronic diseases was gradually discarded. That which had been stated and written by the ancients upon the subject was looked upon as the fictions of an age long past.

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* Vide Epist. XXXVII., on Jaundice.

Modern physiology, however, has effected great changes in this department, and has opened up many new points of view from which to study the question. The secretion of the liver has been carefully examined; the composition, origin and uses of the bile have been closely investigated, and in it, as a constant result of the secreting activity of the gland, sugar has been detected, although the physiological importance of this substance is still obscure. Further, there has been shown to exist in the liver a large quantity of the products of retrograde metamorphosis, which indicate a complicated transformation of materials within the organ, the extent and importance of which, however, still remain to be determined. Lastly, observations have been made which seem to speak in favour of a direct participation of the liver in the formation of the blood.

The task of proving and turning to account these new views will belong to pathology; but there are many difficulties which stand in the way of its performance. Quite independently of the defects which are exhibited almost everywhere in the physiology of the organ before us, and which are sometimes of so important a nature that great caution is necessary in applying our knowledge of its physiology to the investigation of its pathology, the careful study of liver-diseases is impeded by the concealed situation of the organ, by the fact of its secretion being poured high up into the intestinal canal, by the connection of the organ with those parenchymatous changes, the products of which do not make their appearance directly in the excretions, and by the frequent coexistence of diseases of the liver with diseases of other organs which exercise an influence over sanguification and the metamorphosis of matter, such as the digestive organs, the spleen, &c.

In the present work, therefore, many questions are merely announced in a preliminary manner, without our being able to solve them, while others, again, receive only a fragmentary solution. Our investigations, however, on this subject, promise to be not without results, provided we pay attention not merely to anatomical lesions, but also to the disturbances in the metamorphosis of matter by which these lesions are accompanied.

A few references to the most important monographs on Diseases of the Liver are here annexed:—

J. B. BIANCHI. Historia hepatica seu Theoria et praxis omnium morborum hepatis et bilis.—Tom. I. et II.

FR. HOFFMANN. De morbis hepatis ex anatomia deducendis. Also: De bile medicina et veneno corporis. Opera omnia phys. med.—Tom. V.

J. ANDRÉE. Considerations on Bilious Diseases and some particular affections of the Liver and Gall-bladder. London, 1790.

SAUNDERS. A Treatise on the Structure, Economy, and Diseases of the Liver. German translation. Leipsig, 1795.

A. PORTAL. Observations sur la nature et le traitement des maladies du foie. Paris, 1813.

J. ABERCROMBIE. On Diseases of the Stomach, &c. Edinburgh. German translation by Busch, 1833.

BONNET. Traité des maladies du foie. Paris, 1828 and 1841.

G. BUDD. On Diseases of the Liver. London, 1845 and 1851. German edition by Henoch.

The works, moreover, which treat of diseases of the tropics are of importance, especially in reference to inflammation of the liver; such as:—

ANNESLEY. Researches into the cause, nature, and treatment of the more prevalent diseases of India. Vol. I., II., with plates.

CAMBAY. Traité des maladies des pays chauds, et spécialement de l'Algérie. Paris, 1847.

HASPEL. Maladies de l'Algérie. 1852. Tom. II.

CHAPTER II.

THE RELATIVE SIZE AND WEIGHT OF THE LIVER IN HEALTH AND IN DISEASE.

In order to obtain positive data for the solution of many questions, I have deemed it necessary to ascertain the size and weight of the liver, both absolutely and in relation to the weight of the entire body, with greater precision than has hitherto been done. We are dependent upon such knowledge, not merely in a diagnostic point of view, for a correct scale from which to judge of the existence of abnormalities in the size of the organ, such as atrophy, &c.; but we are still more dependent upon it for information as to the manner in which general and local diseases react upon the liver, and as to the extent to which this organ, under the different conditions of the system (resulting from age, sex, and certain diseases), participates in the changes of the nutritive principles.

It would of course have been preferable to have arrived at a satisfactory elucidation of such obscure and complicated questions without having recourse to the *force brutale des chiffres*, yet by such means we may expect to obtain some precise data on which to base further investigation.

It is necessary to take the spleen into consideration at the same time as the liver, partly on account of its intimate relation with the organ secreting bile, and partly on account of the changes in volume of this gland which accompany the different species of affections of the liver, and which are of such importance in the diagnosis of obstructions of the vena portæ.

The determination of the value of figures, as applicable to the purposes just mentioned, is a matter of no small difficulty, inasmuch as, even under normal circumstances, the liver exhibits remarkable differences, which, to a certain extent, cannot easily be accounted for. Hence it is not to be wondered at, that authors should have made widely different statements as to both the absolute* and the relative weight of the liver in healthy individuals. Bartholin estimated the relative weight of the liver in proportion to that of the body as 1 to 36; Haller, as 1 to 25: the average weight of the gland was reckoned by Haller at 45 ounces, or 3.7 pounds, or 1.8 kilogramme; by Cruveilhier, at 3 pounds; and by Huschke at from 4 to 6 pounds. According to my experience, the relative weight of the liver in healthy individuals may vary from one-seventeenth to one-fiftieth of that of the body, and in adults it fluctuates between one-twenty-fourth and one-fortieth; the absolute weight at this period of life, reaches from 0.82 to 2.1 kilogrammes (1.8 to 4.6 English pounds avoird.)* The observations from which these numbers have been derived, were made upon individuals who had died suddenly from accidents without losing any blood, and whose livers, on careful examination, appeared to be healthy. Cases in which there was abundant fatty deposit in the gland were excluded.

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* The absolute weight of the liver usually rises and falls in proportion

There are, therefore, tolerably wide limits which must be exceeded before we can speak of a simple hypertrophy or atrophy of the liver as a pathological phenomenon. The circumstances upon which these differences in the weight of the liver are dependent, have hitherto been only partially discovered. The most important of them are the following.

1. *Age.*

It is during the first stages of development that the organ is largest in proportion to the size of the entire body; even in the later months of fœtal life, and still more so soon after birth, its relative weight declines more and more towards that which is observed at a more advanced age. It is still undetermined, whether the remarkable diminution observed during fœtal life is of a uniform nature; many observations are opposed to such being the case. After birth, the entire gland, but especially the left lobe,† dimi-

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to the weight of the body, so that the term absolute can only be employed in a comparative sense.

* Similar differences were observed by Bidder and Schmidt (*Verdauungsäfte, etc., S.* 152), in healthy animals, such as cats, in which the ratio was from as 1 to 14, to 1 to 38.

† According to Portal and Meckel the liver in newly-born children ought to be one-fourth heavier than in children from eight to ten months old; but my experience does not correspond with this.

nishes more rapidly in consequence of the altered supply of blood. During the period of greatest growth, the liver does not become enlarged in a manner proportionate to the increase of the entire body, and its diminution in old age is, for the most part, in advance of that of the body. The substance of the liver, therefore, presents in this respect, a marked contrast to the muscular tissue of the heart, for whilst this, according to Bizot, increases progressively up to an advanced age, the mass of the liver diminishes. In old age, as a general rule, there is senile atrophy of the organ.

Such are the results of weighing the liver in healthy individuals, as well as in numerous instances of persons who have died of other diseases in which the liver has not been at all involved. Exceptions occur, owing to the force of circumstances of an extraneous nature influencing the volume of the gland; but these exceptions disappear on adding together the larger series of figures and calculating the average.*

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* The number of original observations as to the weight and size of the liver from which my calculations have been made, amounts to about 800. A large number of weighings appeared necessary, because numerous accidental influences, modifying the weight of the liver as well as of the entire body, such as hyperæmia and anæmia of the organ, dropsy, suppression, or profuse secretion of bile must be excluded. Space does not permit us to give here a detailed account of the numbers in each case. In the tables the measurements are indicated by Paris inches; the weights, by kilogrammes.

TABLE I.

RELATIVE AND ABSOLUTE WEIGHT, ETC., OF THE LIVER UNDER NORMAL CONDITIONS.*

Age.	Weight		Relative Weight of Liver to that of Body.	Weight of Spleen.	Relative weight of Spleen to that of		Dimensions of Liver.					Dimensions of Spleen.		
							Length.		Breadth.		Thickness (greatest).	Length.	Breadth.	Thickness.
	of Body.	of Liver.			Body.	Liver.	Right Lobe.	Left Lobe.	Right Lobe.	Left Lobe.				
	Kilogr.	Kilogr.		Kilogr.			Par. in.	Par. in.	Par. in.	Par. in.	Par. in.	Par. in.	Par. in.	Par. in.
5 months Fœtus	0.72	0.035	1:20.5	0.0025	1:288.0	1:14.0	—	—	—	—	—	—	—	—
6 " "	1.3	0.060	1:21.6	0.004	1:325.0	1:15.	2½	2	2	1¾	⅔	⅝	7/12	⅓
7 " "	2.2	0.13	1:17.0	0.006	1:366.6	1:21.6	3	2⅝	2	2½	1	1½	11/12	5/12
New-born Child	1.6	0.056	1:28.57	0.008	1:200.0	1: 7.	2½	2	2	1¼	1	1⅛	1	⅓
Do. do.	1.4	0.058	1:24.1	0.011	1:145.54	1: 5.27	2¼	1¾	2½	1¼	11/12	1¼	1	½
1 day old	3.8	0.185	1:20.5	0.011	1:345.4	1:16.8	2¾	2½	2¾	2	1 1/12	1½	1	5/12
8 days	2.7	0.103	1:26.1	0.009	1:248.6	1:11.	2½	2¼	2⅓	1¾	11/12	1¾	1⅛	5/12
5 weeks	1.95	0.090	1:21.66	0.016	1:121.8	1: 5.62	2½	2	1	1⅓	1	2	1⅓	½
15 months	8.3	0.25	1:33.2	0.020	1:415.0	1:12.5	3½	4	3½	1¾	1½	2½	1¼	½
5 years	8.8	0.48	1:18.3	0.1	1: 88.0	1: 4.8	3½	4¾	4¾	2¾	1½	3½	2¼	1
11 "	24.8	0.97	1:25.56	0.14	1:177.14	1: 6.9	6¼	3	6¼	3½	3¼	4½	3	¾
22 "	64.5	1.6	1:40.3	0.16	1:403.	1:10.	—	—	—	—	—	—	—	—
27 "	50.0	1.9	1:26.5	0.22	1:227.	1: 8.6	—	—	—	—	—	—	—	—
35 "	32.0	0.82	1:39.0	0.08	1:400.	1:10.2	6	4¼	5¼	2½	2	3¾	2½	½
36 "	55.5	1.5	1:37.0	0.15	1:370.	1:10.	7	5½	5½	4	2¼	4	2¾	¾
44 "	56.2	1.4	1:40.1	0.25	1:224.8	1: 5.6	7¼	5¼	6	3¼	2¼	5	3¼	1¼
49 "	59.5	1.47	1:40.1	0.15	1:390.	1: 9.8	7¼	4¾	6⅕	3¼	2¼	5¼	3	⅔
63 "	45.5	1.4	1:32.5	0.12	1:379.	1:11.6	7⅓	5½	6	3	2½	5	3	¾
80 "	30.1	0.7	1:43.5	0.1	1:305.	1: 7.	—	—	—	—	—	—	—	—

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* A kilogramme is about 2¼, or, more correctly, 2.2046 pounds avoird.; the weight in English pounds avoird., will therefore be ascertained by multiplying the numbers in the table by 2.2046.

One Paris inch contains 12 Paris lines, but an English inch only 11.25 Paris lines; hence the measurements in English inches will be slightly greater than those given in the table, or may be ascertained by dividing the numbers in the table by .9375.—TRANSL.

2. *The Sex.*

According to Glisson, the liver, as a general rule, is heavier in men than in women; but an opinion precisely the reverse is maintained by Dumas. I myself have been unable to detect any differences dependent upon sex, except that in scrofulous women the liver is usually larger than in men, from the fact, that in the former sex, it is more frequently remarkable for an abundant deposit of fatty matter.

3. *The Ingestion of Food*

exerts a powerful influence over the volume of the liver. During the second stage of digestion the organ increases in size and weight, partly owing to the state of congestion which then makes its appearance, and partly owing to the abundant deposit of granular and amorphous material in the interior of the hepatic cells. After a protracted fast the gland becomes smaller and lighter. Bidder and Schmidt found that in cats the relative weight of the liver to that of the entire body was as 1 to 30, three hours after a meal; after from twelve to fifteen hours, it was 1 to 25; from twenty-four to forty-eight hours after, it was 1 to 31; and after seven days' fast, 1 to 37. I have arrived at similar results in rabbits; under the influence of a three days' fast, the relative weight, which after a full meal is, generally, 1 in 25, or 1 in 27, diminished to 1 in 34, 1 in 37, and 1 in 43, with a total loss in each case of 31.1, 29.6, and 17.8 per cent. The following observations appear to indicate that starvation is followed by similar consequences in men. In a healthy individual aged 27, who, owing to a fall from a scaffold, died with a full stomach, the relative weight of the liver was found to be as 1 to 26.5; in another person aged 36, who perished under similar circumstances, it was as 1 to 37; on the other hand, in a man aged 25, who died of trismus, after three days' complete abstinence, it was 1 to 40; and in a woman, 33 years of age, who died from burning the pharynx with sulphuric acid, after seven days' fasting, it was 1 to 50.

From what has just been stated it may be concluded, that, in the management of chronic congestions of the liver, a strict diet plays a powerful part in effecting a cure. In cases, however, of protracted starvation, which terminate in death from inanition, the diminution of the liver in proportion to that of the entire body, becomes again equalized. Thus, the following observations were made in four individuals who died of stricture of the œsophagus.

Age.	Weight		Relative weight of Liver to that of Body.	Weight of the Spleen.	Relative weight of Spleen to that of	
	Of the Body.	Of the Liver.			Liver.	Body.
	Kilogr.[*]	Kilogr.		Kilogr.		
48 years .	32.2	1.1	1 : 29.2	0 : 13	1 : 8.4.	1 : 247
50 " .	30.8	0.92	1 : 33.47	—	—	—
65 " .	39.0	1.20	1 : 32.5	0 : 12	1 : 10.	1 : 325
44 " .	39.7	1.75	1 : 22.7	—	—	—

Still more remarkable is the influence of diet, when the food is very rich in fat, or when it is too bulky, while at the same time the powers of digestion are impaired. In this case the deposit of fat in the hepatic parenchyma induces an undue proportion in the size of the liver. Bidder and Schmidt found the relative weight under such circumstances to be as 1 to 16. Lereboullet ascertained, that in geese the relative weight of the liver varied from 1 in 26 to 1 in 18, after feeding for two weeks upon maize, and that after four weeks it rose to 1 in 12.8.

How greatly the volume of the liver is influenced by nourishment may easily be seen by comparing a large number of observations on the weight of the organ in individuals who have died of pneumonia, typhus, and other febrile complaints. Its relative size under such circumstances is much smaller and more uniform than what it is when death has not been preceded by protracted starvation. (See Table II.)

4. *The Degree of Congestion of the Liver*

has a marked influence over its size and weight; changes result from this cause, which are of little importance, because the causes of the abnormal distribution of the blood have in a measure only a transitory and accidental value. We are possessed of no means for completely comprehending the derangements which result from this cause, whilst the attempts to separate the blood before weighing, by the injection of water have induced other sources of fallacy.

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[*] 1 Kilogr. = 2.2046 English pounds avoird.

It is not improbable, that in addition to the influences just mentioned, there are still others which operate in changing the volume of the liver, so that the organ will be found sometimes exceeding, and sometimes smaller than, its normal proportions, according to the constitutional peculiarities of the individual.

No certain proofs, however, can be furnished of the existence of such agencies, because we know not the limits to which other causes, which are at least partially understood, modify the size of the organ, and consequently, in some cases, we are not in a position to exclude these causes.

The Relative Weight and Size of the Liver in Disease.

In order to obtain an insight into the manner in which the relative volume of the liver becomes affected in the course of general and local diseases, and as to how the organ becomes altered in size by morbid conditions of its own textures, several special series of measurements have been collected. The most important results of these measurements have been noted in the annexed tables. These observations furnish us with trustworthy data for the solution of many questions, and their value will appear in a subsequent part of this Work.

TABLE II.

WEIGHT AND DIMENSIONS OF THE LIVER IN ACUTE DISEASES IN WHICH THE ORGAN ITSELF IS NOT DIRECTLY INVOLVED.

A. MALES.

Disease.	Age. Between	Number of Cases.	Weight of		Relative Weight of Liver to that of Body.	Weight of Spleen.	Relative Weight of Spleen to that of		Dimensions of Liver in Paris inches.					Dimensions of Spleen in Paris inches.		
									Length.		Breadth.		Thickness (greatest).			
			Body.	Liver.			Body.	Liver.	Right Lobe.	Left Lobe.	Right Lobe.	Left Lobe.		Length.	Breadth.	Thickness.
			Kilogr.	Kilogr.		Kilogr.			Par. in.	Par. in.	Par. in.	Par. in.	Par. in.	Par. in.	Par. in.	Par. in.
Pneumonia	15 & 20	2	35.5	1.33	1.26.4	0.45	1:173.4	1:6.5	$6\frac{1}{2}$	$5\frac{1}{4}$	$6\frac{5}{8}$	$3\frac{1}{4}$	$2\frac{1}{3}$	$5\frac{1}{2}$	$3\frac{5}{8}$	$1\frac{1}{8}$
	20 „ 40	13	49.76	1.82	1:28.3	0.235	1:270.7	1:9.37	$7\frac{2}{5}$	$5\frac{1}{3}$	$6\frac{1}{3}$	$3\frac{1}{2}$	$2\frac{1}{2}$	$5\frac{2}{3}$	$3\frac{1}{10}$	$1\frac{1}{12}$
	40 „ 50	12	55.0	2.05	1:27.72	0.32	1:192.2	1:7.1	$7\frac{1}{8}$	$5\frac{5}{6}$	$7\frac{3}{8}$	$3\frac{1}{8}$	$2\frac{7}{8}$	$5\frac{5}{8}$	$3\frac{3}{8}$	$1\frac{1}{8}$
	50 „ 60	4	43.5	1.65	1:26.2	0.2	1:259.4	1:10.2	$7\frac{1}{8}$	$5\frac{1}{2}$	$6\frac{1}{2}$	$3\frac{1}{3}$	$2\frac{3}{4}$	5	$3\frac{1}{4}$	$\frac{7}{8}$
	60 „ 80	11	47.7	1.66	1:29.2	0.23	1:226.0	1:7.4	7	$5\frac{1}{3}$	$6\frac{1}{3}$	$3\frac{3}{4}$	$2\frac{1}{3}$	$5\frac{1}{4}$	$3\frac{1}{4}$	$1\frac{1}{10}$
		42			1:28.4		1:241.9	1:8.6								
Typhus	7 „ 20	9	37.6	1.34	1:27.15	0.34	1:104.1	1:3.87	$6\frac{3}{8}$	$5\frac{1}{3}$	6	3	$2\frac{1}{4}$	6	$3\frac{3}{8}$	$1\frac{1}{8}$
	21 „ 60	20	46.93	1.63	1:28.8	0.45	1:124.7	1:4.3	$7\frac{1}{3}$	$5\frac{3}{4}$	$6\frac{1}{4}$	$3\frac{1}{8}$	$2\frac{1}{2}$	$6\frac{1}{4}$	$3\frac{3}{4}$	$1\frac{1}{3}$
		29			1:28.66		1:122.75	1:4.24								
Acute Miliary Tubercle	22 „ 60	3	43.6	1.36	1:32.4	0.256	1:180.1	1:5.53	$7\frac{1}{6}$	$5\frac{1}{2}$	$5\frac{3}{4}$	3	$2\frac{5}{8}$	$5\frac{2}{3}$	$3\frac{1}{8}$	$1\frac{1}{4}$
Pyæmia	17 „ 45	3	47.2	1.55	1:31.1	0.24	1:192.5	1:6.4	$7\frac{1}{3}$	5	6	$3\frac{3}{4}$	$2\frac{1}{2}$	5	$3\frac{1}{4}$	$1\frac{1}{4}$
Variola	36 „ 62	3	46.06	1.73	1:26.4	0.15	1:299.3	1:11.3	$8\frac{3}{8}$	$5\frac{3}{8}$	$5\frac{3}{8}$	$3\frac{3}{8}$	$2\frac{3}{8}$	$4\frac{1}{3}$	$2\frac{1}{4}$	1
Acute Bright's Disease	30 „ 62	3	52.16	1.55	1:33.9	0.26	1:207.4	1:6.4	7	$5\frac{7}{8}$	$6\frac{3}{4}$	$2\frac{1}{3}$	$2\frac{1}{3}$	$5\frac{3}{4}$	$3\frac{1}{2}$	$\frac{3}{4}$
Acute Peritonitis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

B. FEMALES.

Disease.	Age. Between	Number of Cases.	Weight of		Relative Weight of Liver to that of Body.	Weight of Spleen.	Relative Weight of Spleen to that of		Dimensions of Liver, in Paris inches.					Dimensions of Spleen in Paris inches.		
									Length.		Breadth.					
			Body.	Liver.			Body.	Liver.	Right Lobe.	Left Lobe.	Right Lobe.	Left Lobe.	Thickness.	Length.	Breadth.	Thickness.
			Kilogr.	Kilogr.		Kilogr.										
Pneumonia	— —	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	20 & 40	5	36.06	1.41	1:27.04	0.23	1:210.14	1: 7.86	7¾	4 9/10	5⅓	3½	2⅔	4⅓	3⅓	1
	— —	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	50 „ 60	4	40.7	1.4	1:29.8	0.19	1:223.12	1: 7.42	7	5¼	5½	3	2¾	4⅞	3	1
	60 „ 80	11	39.8	1.39	1:29.4	0.18	1:248.3	1: 8.5	7	5⅓	5 9/11	3¾	2½	4 7/11	2 9/11	8/11
		20			1:29.0		1:232.1	1: 8.05								
Typhus	17 „ 20	3	39.9	1.72	1:25.8	0.25	1:178.7	1: 8.0	7	5½	5⅝	3		5	3½	1⅛
	21 „ 50	10	41.16	1.44	1:28.45	0.35	1:120.67	1: 4.25	7½	5¼	6⅓	4	2¼	6⅔	3½	1¼
		13			1:27.72		1:140.4	1: 5.36								
Acute Miliary Tubercle	— „ 30	1	26.2	1.4	1:18.7	0.3	1: 87.3	1: 4.6	8¼	5½	6	3¾	2	5½	3¾	1½
Pyæmia	25 „ 49	2	43.8	1.56	1:27.9	0.35	1:136.2	1: 3.0	6¾	6	5¾	4	2½	6	3½	1¼
Variola	28 „ 35	2	43.2	1.65	1:26.2	0.14	1:302.7	1:12.24	7	5½	5⅞	3	2	3¼	2	1
Acute Bright's Disease	— —	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Acute Peritonitis	33 „ 84	5	41.9	1.27	1:32.9	0.13	1:340.1	1:10.5	7½	4¾	6	3	2⅓	4½	2¾	⅞

TABLE III.

WEIGHT AND DIMENSIONS OF THE LIVER IN CHRONIC DISEASES IN WHICH THE ORGAN ITSELF IS NOT DIRECTLY INVOLVED.

A. MALES.

Diseases.	Age. Between	Number of Cases.	Weight of		Relative Weight of Liver to that of Body.	Weight of Spleen.	Relative Weight of Spleen to that of		Dimensions of Liver in Paris inc.					Dimensions of Spleen in Paris inc.		
									Length.		Breadth.					
			Body.	Liver.			Body.	Liver.	Right Lobe.	Left Lobe.	Right Lobe.	Left Lobe.	Thickness.	Length.	Breadth.	Thickness.
			Kilogr.	Kilogr.		Kilogr.										
Tubercle	10 & 20	4	20.6	0.81	1:24.9	0.10	1 :212.2	1: 8.6	$5\frac{7}{16}$	$4\frac{11}{16}$	$4\frac{11}{16}$	$2\frac{15}{16}$	$2\frac{1}{4}$	$3\frac{7}{8}$	$2\frac{1}{2}$	$\frac{5}{8}$
	20 „ 40	45	41.3	1.47	1:23.7	0.23	1:193.8	1: 6.5	$7\frac{1}{14}$	$5\frac{1}{8}$	$6\frac{1}{14}$	$3\frac{5}{9}$	$2\frac{1}{3}$	$5\frac{1}{5}$	$3\frac{1}{5}$	$1\frac{1}{10}$
	40 „ 60	30	44.56	1.42	1:31.23	0.198	1:270.84	1: 8.1	$6\frac{5}{6}$	$5\frac{1}{8}$	6	$3\frac{1}{2}$	$2\frac{9}{16}$	$4\frac{11}{12}$	$2\frac{15}{16}$	$1\frac{1}{6}$
	60 „ 80	7	40.3	1.19	1:34.6	0.19	1:227.1	1: 6.6	$6\frac{2}{7}$	$5\frac{3}{4}$	$5\frac{1}{2}$	$3\frac{1}{14}$	$2\frac{1}{4}$	$4\frac{1}{2}$	$2\frac{4}{5}$	$1\frac{1}{7}$
		86			1:30.0		1:224.17	1: 7.3								
Emphysema of Lungs	50 „ 80	3	38.7	1.30	1:29.9	0.16	1:258.8	1: 8.5	$6\frac{5}{6}$	5	$5\frac{2}{3}$	3	$2\frac{1}{4}$	4	$2\frac{1}{3}$	$1\frac{1}{2}$
Organic Disease of Heart—																
A. Without Dropsy	19 „ 70	13	50.4	1.58	1:35.66	0.23	1:276.11	1: 7.5	7	5	$5\frac{11}{15}$	$3\frac{1}{2}$	$2\frac{1}{5}$	$4\frac{3}{4}$	3	$1\frac{1}{4}$
B. With Dropsy	— „ 38	1	75.0	1.5	1:50.	0.19	1:394.0	1: 8.0	$7\frac{3}{4}$	5	$6\frac{1}{2}$	$3\frac{1}{4}$	$2\frac{3}{4}$	$4\frac{3}{4}$	3	$\frac{3}{4}$
Carcinoma—																
A. Without Dropsy	50 „ 60	4	39.55	0.92	1.43.7	0.128	1:317.1	1: 7.07	$5\frac{13}{16}$	$4\frac{1}{8}$	$5\frac{9}{16}$	$3\frac{11}{16}$	$2\frac{1}{16}$	$4\frac{5}{8}$	$2\frac{3}{4}$	1
B. With Dropsy	— „ 59	1	48.07	1.37	1:35.1	0.14	1:343.3	1: 9.6	$6\frac{1}{2}$	$4\frac{1}{2}$	$5\frac{1}{2}$	3	$3\frac{1}{4}$	4	$2\frac{3}{4}$	1
C. With Stricture of Œsophagus (Inanition)	48 „ 80	5	37.3	1.23	1:30.6	0.12	1:378.2	1:12.4	7	5	$5\frac{3}{10}$	3	$2\frac{3}{10}$	$3\frac{17}{20}$	$2\frac{13}{20}$	$\frac{4}{5}$
Delirium tremens	28 „ 62	10	56.8	2.05	1:27.2	0.30	1:189.9	1: 6.8	$7\frac{1}{2}$	$5\frac{3}{4}$	5	$3\frac{1}{3}$	3	$5\frac{1}{2}$	$3\frac{1}{4}$	$\frac{1}{3}$

B. FEMALES.

Diseases.	Age. Between	Number of Cases.	Weight of Body.	Weight of Liver.	Relative Weight of Liver to that of Body.	Weight of Spleen.	Relative Weight of Spleen to that of Body.	Relative Weight of Spleen to that of Liver.	Dimensions of Liver in Paris inc. Length. Right Lobe.	Length. Left Lobe.	Breadth. Right Lobe.	Breadth. Left Lobe.	Thickness.	Dimensions of Spleen in Paris inc. Length.	Breadth.	Thickness.
Tubercle	10 & 20	5	Kilogr. 23.6	Kilogr. 1.13	1:21.06	Kilogr. 0.18	1:156.6	1: 7.7	$6\frac{3}{5}$	$5\frac{3}{20}$	$5\frac{13}{20}$	$3\frac{3}{5}$	$2\frac{3}{4}$	$4\frac{13}{20}$	$2\frac{3}{4}$	1
	20 „ 40	30	34.8	1.35	1:25.45	0.196	1:193.24	1: 7.54	$7\frac{1}{8}$	$5\frac{1}{2}$	$5\frac{1}{2}$	$3\frac{3}{8}$	$2\frac{1}{7}$	5	$2\frac{5}{8}$	$1\frac{1}{3}$
	40 „ 60	16	33.8	1.17	1:28.6	0.14	1:260.27	1: 8.94	$7\frac{9}{16}$	$5\frac{9}{16}$	$5\frac{9}{16}$	$3\frac{3}{8}$	$2\frac{1}{16}$	$4\frac{7}{16}$	$2\frac{3}{8}$	$\frac{15}{16}$
	60 „ 80	10	32.48	0.252	1:36.02	0.102	1:383.3	1:10.23	$6\frac{7}{8}$	$5\frac{1}{2}$	5	$2\frac{6}{7}$	$1\frac{7}{8}$	$3\frac{11}{13}$	$2\frac{3}{8}$	$\frac{4}{5}$
		61			1:28.53		1:245.8	1: 8.61								
Emphysema of Lungs	50 „ 80	6	39.6	1.18	1:35.4	0.23	1:242.1	1: 6.04	$6\frac{5}{6}$	$4\frac{1}{2}$	$5\frac{2}{3}$	$3\frac{3}{4}$	$2\frac{1}{12}$	$4\frac{1}{6}$	$2\frac{1}{12}$	$1\frac{1}{24}$
Organic Disease of Heart—																
A. Without Dropsy	20 „ 80	9	41.3	1.22	1:37.9	0.17	1:282.5	1: 8.09	$6\frac{5}{6}$	$4\frac{8}{9}$	$5\frac{1}{2}$	$3\frac{1}{4}$	$2\frac{1}{4}$	$4\frac{2}{3}$	$2\frac{1}{3}$	$\frac{11}{12}$
B. With Dropsy	25 „ 80	13	57.3	1.29	1:47.6	0.14	1:464.03	1: 9.3	$7\frac{1}{6}$	$5\frac{1}{6}$	$5\frac{3}{8}$	3	$2\frac{3}{10}$	$3\frac{12}{13}$	$2\frac{10}{13}$	$1\frac{1}{13}$
Carcinoma—																
A. Without Dropsy	40 „ 80	13	39.4	1.1	1:37.2	0.13	1:341.3	1: 9.79	7	$5\frac{1}{2}$	$5\frac{1}{2}$	$3\frac{1}{2}$	$2\frac{1}{6}$	$4\frac{1}{4}$	$2\frac{7}{12}$	$\frac{11}{12}$
B. With Dropsy	30 „ 60	6	50.6	1.23	1:41.25	0.19	1:268.3	1: 6.43	$6\frac{11}{12}$	$5\frac{1}{6}$	$5\frac{2}{3}$	$3\frac{1}{4}$	$2\frac{1}{2}$	$4\frac{1}{2}$	3	$1\frac{1}{4}$
C. With Stricture of Œsophagus (Inanition)	— —	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Delirium tremens	— —	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

TABLE IV.

WEIGHTS, DIMENSIONS, ETC., OF THE LIVER, IN DISEASED STATES OF THE ORGAN.

A. MALES.

Diseases.	Age. Between	Number of Cases.	Weight of		Relative Weight of Liver to that of Body.	Weight of Spleen.	Relative Weight of Spleen to that of		Dimensions of Liver in Paris inc.					Dimensions of Spleen in Paris inc.		
									Length.		Breadth.					
			Body.	Liver.			Body.	Liver.	Right Lobe.	Left Lobe.	Right Lobe.	Left Lobe.	Thickness.	Length.	Breadth.	Thickness.
			Kilogr.	Kilogr.		Kilogr.										
Acute Atrophy	— —	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Chronic Atrophy—																
A. Without Dropsy	53 & 64	4	41.2	0.81	1:53.8	0.168	1:344.5	1:5.9	$5\frac{13}{16}$	$3\frac{3}{4}$	$5\frac{1}{8}$	$2\frac{7}{8}$	$1\frac{4}{5}$	$4\frac{5}{8}$	3	$\frac{1}{2}$
B. With Dropsy	35 „ 58	4	61.3	0.56	1:71.0	0.18	1:360.6	1:5.29	$5\frac{5}{8}$	$5\frac{3}{5}$	$5\frac{1}{2}$	$4\frac{5}{16}$	$1\frac{3}{8}$	$5\frac{1}{8}$	$3\frac{3}{16}$	1
Cirrhosis of Liver—																
A. Without Dropsy	20 „ 71	9	44.84	1.693	1:32.13	0.21	1:270.55	1:7.9	$6\frac{11}{12}$	$5\frac{2}{3}$	$5\frac{4}{9}$	$3\frac{3}{8}$	$2\frac{3}{8}$	$4\frac{11}{12}$	$3\frac{1}{8}$	$1\frac{1}{8}$
B. With Dropsy	30 „ 59	3	73.0	1.76	1:42.2	0.58	1:139.7	1:4.03	$5\frac{5}{6}$	$5\frac{5}{6}$	$5\frac{7}{12}$	$4\frac{1}{3}$	$2\frac{3}{8}$	$7\frac{1}{12}$	$4\frac{7}{12}$	$1\frac{1}{2}$
Waxy Liver—																
A. Without Dropsy	18 „ 24	2	31.1	1.37	1:24.0	0.267	1:130.1	1:5.2	$6\frac{1}{2}$	$4\frac{3}{4}$	$5\frac{5}{8}$	$3\frac{3}{8}$	$2\frac{3}{4}$	$5\frac{1}{4}$	$3\frac{1}{4}$	$1\frac{5}{8}$
B. With Dropsy	— „ 57	1	73.0	1.18	1:61.18	0.46	1:158.6	1:2.5	$6\frac{1}{4}$	$2\frac{1}{2}$	$5\frac{3}{4}$	$4\frac{1}{2}$	$2\frac{3}{4}$	$6\frac{1}{4}$	4	$2\frac{1}{4}$
Pigment Liver in Intermittent Fever	22 „ 45	2	66.7	1.61	1:41.9	0.71	1: 94.4	1:2.2	$6\frac{5}{8}$	$5\frac{3}{4}$	$6\frac{3}{4}$	$3\frac{1}{4}$	$2\frac{1}{2}$	$7\frac{1}{4}$	5	2
Fatty Liver	29 „ 68	18	47.6	1.68	1:28.55	0.22	1:247.8	1:8.51	$7\frac{2}{3}$	$5\frac{1}{2}$	$5\frac{4}{5}$	$3\frac{1}{5}$	$2\frac{5}{8}$	$4\frac{1}{3}$	3	$1\frac{1}{3}$
Retention of Bile	36 „ 55	3	45.8	1.48	1:33.2	0.3	1:196.9	1:4.13	$7\frac{7}{8}$	$5\frac{3}{4}$	6	$3\frac{5}{8}$	$2\frac{1}{8}$	6	$4\frac{1}{4}$	$1\frac{3}{4}$
Carcinoma of Liver	41 „ 54	3	53.4	3.16	1:21.2	0.22	1:249.8	1:4.2	$8\frac{1}{2}$	$5\frac{11}{12}$	$7\frac{1}{6}$	4	$3\frac{5}{12}$	$4\frac{1}{2}$	3	$1\frac{1}{6}$
Diabetes mellitus	— —	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

B. FEMALES.

Diseases.	Age. Between	Number of Cases.	Weight of		Relative Weight of Liver to that of Body.	Weight of Spleen.	Relative Weight of Spleen to that of		Dimensions of Liver in Paris inc.					Dimensions of Spleen in Paris inc.		
									Length.		Breadth.					
			Body.	Liver.			Body.	Liver.	Right Lobe.	Left Lobe.	Right Lobe.	Left Lobe.	Thickness.	Length.	Breadth.	Thickness.
			Kilogr.	Kilogr.		Kilogr.										
Acute Atrophy - - - -	— & 24	1	56.2	0.82	1:68.5	0.37	1:151.9	1: 2.2	5½	5¼	5¼	3	1⅛	5½	3¾	1⅛
Chronic Atrophy—																
A. Without Dropsy -	50 „ 78	3	30.4	0.68	1:44.76	0.096	1:358.9	1: 7.8	5⅛	4 5/12	4⅛	3	1¾	3⅝	2⅛	⅝
B. With Dropsy - - -	— „ 56	1	50.2	0.95	1:52.9	0.08	1:627.0	1:11.8	6¾	5	5½	2½	2¼	4	2¼	3
Cirrhosis of Liver—																
A. Without Dropsy -	34 „ 77	8	41.92	1.38	1:31.7	0.17	1:269.15	1: 8.62	8 13/16	6 1/10	5⅛	3 3/16	2⅓	4 5/16	3	⅔
B. With Dropsy - - -	27 „ 58	3	53.76	0.928	1:60.1	0.12	1:541.95	1: 9.0	6 11/12	4 11/12	4¾	2 19/36	2⅓	4	2 7/12	⅚
Waxy Liver—																
A. Without Dropsy -	— —	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
B. With Dropsy - - -	14 „ 42	4	55.0	1.31	1:42.9	0.248	1:256.0	1: 6.0	7 3/16	4⅝	5 1/7	2¾	2 3/16	4 15/16	3 1/16	1 5/16
Pigment Liver in Intermittent Fever - - -	— —	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Fatty Liver - - - - - -	17 „ 84	16	36.8	1.5	1:25.7	0.24	1:183.7	1: 7.58	7⅜	5⅝	6⅛	3⅛	2⅛	5¼	3⅛	1 3/16
Retention of Bile - - -	— —	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Carcinoma of Liver - -	31 „ 72	2	38.1	1.94	1:21.47	0.17	1:223.3	1:11.1	8	6⅝	6¾	4	2¾	5⅜	2¾	1
Diabetes mellitus - - -	— „ 37	1	30.3	1.4	1:23.5	0.21	1:157.1	1: 6.6	8¼	6¼	5½	3	2⅓	5¾	2¾	1

CHAPTER III.

A SKETCH OF THE RELATIVE SIZES AND FORMS OF THE LIVER IN ITS MORBID CONDITIONS, AND OF THEIR DIAGNOSTIC VALUE.

In the diagnosis of diseases of the liver, an accurate knowledge of the size and form of the organ is one of the first points to be ascertained. Such information furnishes valuable data, which in some cases are of themselves sufficient for the diagnosis, and which in most enables us to refer the disease to a certain group, so as to narrow the field of choice. The positive data which we obtain by this means are always of great value, although we cannot draw any conclusions as to the healthy condition of the organ, from the fact of its form and size being normal. Thus, there may exist deep-seated diseases of the parenchyma of the liver, although on physical examination, no deviation of its external characters can be detected.* Not only functional derangements and the more minute alterations of tissue, but also the coarser anatomical lesions, such as cancer, echinococci, &c., may not unfrequently attain a considerable development in the interior of the liver, without modifying in a marked degree its volume and form. As one of many similar instances, I may mention one where a large sac of an echinococcus was situated in the right lobe of the liver underneath the diaphragm, and penetrated deeply into the parenchyma without altering the extent or form of the organ in any way capable of assisting diagnosis. A similar condition is not unfrequently met with in the case of cancer; here, indeed, it is sometimes impossible, even after the removal of the organ, to detect upon its surface with closed eyes, and by the sense of touch merely, any nodules existing in its interior.

With some practice it is not difficult, by means of percussion and palpation, to obtain data which may be of service in diagnosis; it is a far less easy matter to turn these data to a proper account. The impediments which here present themselves cannot be overcome by any general rules, but only by an attentive consideration of all the circumstances of the case, and by a careful sifting of the numerous causes of the abnormality in question. Some of these last are owing to the great variety of forms which the healthy liver may exhibit; others, to the very frequent anomalies in the situation and position of this gland; and lastly, a third class are dependent upon the difficulty which there is in distinguishing, by any diagnostic means placed at our command, between the boundaries of the liver, and those of adjoining organs and pathological growths.

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* Even the ancient physicians were familiar with this fact; Baillou (*Consultationes med.* T. II., p. 56) justly observes:—"Hepar non desinit male haberi, etsi nihil fores appareat."

† In the original German edition, a figure is given of the liver here alluded to.—Transl.

The Normal Situation and Size of the Liver.

The liver is situated in the right hypochondrium, filling up the concavity of the diaphragm upon this side, and is almost completely concealed by the arch of the ribs, which usually leave uncovered the part of the left lobe which projects into the epigastrium. Its convex upper surface corresponds to the vault of the diaphragm, and projects into the right side of the thorax, where it is partially separated from the walls of the chest by the intervention of the thin lower margin of the right lung, the greater portion of the organ, however, being in close juxtaposition to these walls. In any investigation, therefore, the upper boundary may be said to be twofold; thus, there is one boundary which corresponds to the limit of the region in which the organ is in close approximation to the walls of the chest, and which is distinguished by a dull percussion-sound; and there is a second, which represents the absolute height of the liver, which corresponds to the space overlapped by the thin layer of lung, and which is recognised by the transition from a muffled sound, on percussion, to the full clear pulmonary note. In healthy individuals, the situation of these upper boundaries is tolerably uniform.

In the median line, at the junction of the ensiform-cartilage with the sternum, the liver is in contact with the walls of the chest, and from this it stretches almost horizontally, yet slightly downwards and backwards, through the right half of the thorax towards the vertebral column.

In the *linea mammalis*, or a line extending perpendicularly downwards from the right nipple, the upper boundary (uncovered by lung) corresponds in most cases to the sixth rib; along the *linea axillaris*, to the eighth; and at the side of the vertebral column to the eleventh. The extent of the space covered by the layer of lung amounts to from two to five, usually three, centimètres (3 cent. =1⅕, or 1.18 English inch), and above this is the true upper margin of the organ; this space is somewhat more extensive in a line with the axilla than in a line with the mamma, and usually it is larger in tall persons than in those of short stature. It follows, that the true upper margin of the liver, along the *linea mammalis*, in most cases, lies in the fifth intercostal space, less frequently beneath the fifth rib or in the fourth intercostal space; in the *linea axillaris* it is in the seventh intercostal space, or more seldom under the seventh rib; and close to the vertebral column, it is in the tenth intercostal space, or less frequently in the ninth.

The upper boundary of the liver at the median line cannot usually be distinguished from the lower margin of the heart.* It extends from three to twelve, but in most cases about seven centimètres (three English inches), to the left beyond the median line. Its upper margin at this part corresponds to the lower margin of the heart, and can only be made out with certainty by percussion, where it projects beyond this organ to the left.

The position of the lower margin is much less constant than that of the upper. Even in healthy persons, it is very variable, owing to the great varieties in the form of the liver, and to abnormalities in the configuration of the lower part of the thoracic walls. In a line with the nipple, the lower edge of the liver is found sometimes to coincide with the lower margin of the ribs, and at other times to be from two to four centimètres (four-fifths to one and three-fifths of an English inch) higher up beneath the ribs; it may, however, extend to seven centimètres (nearly three inches) beyond the lower margin of the ribs, and in certain abnormal forms of the liver, such as in tight-lace-liver (*Schnürleber*), still deeper, without being indicative of any disease. In the *linea axillaris*, the lower margin lies usually in the tenth intercostal space, rarely in the ninth; it may, however, here also project from two to four centimètres beyond the margin of the ribs, and under some circumstances even farther, quite independently of any lesion of the organ. At the side of the vertebral column it is impossible to make out the boundary between the lower margin and the right kidney. The lower edge of the left lobe is from five to fourteen centimètres (two to five and a-half inches) distant from the base of the ensiform cartilage, or more correctly from the line above-mentioned, as corresponding to the lower margin of the heart; in most cases, it lies somewhat higher than the middle of a line drawn from the umbilicus to the apex of the ensiform cartilage.

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* According to the advice given by Conradi, the upper boundary of the liver at this place may be made out most easily, by drawing a straight line from the point of contact of the right margin of the cardiac dulness with the upper boundary of the liver to the apex of the cardiac dulness on the left.

The lower margin as far as the left extremity of the organ is more or less rounded, according to the form of the left lobe, which is very variable. Hence, to sum up, the position of the lower margin of the liver, and its relation to the edge of the thoracic walls vary so greatly in healthy individuals, that the greatest caution is required in making use of these signs in diagnosis. It is, therefore, only permissible to draw any conclusion as to the enlargement or dislocation of the organ, from the mere fact of its protruding beyond the margin of the walls or the thorax, when the extent of this protrusion is considerable.

The statements just made as to the relative situations of the liver have mostly reference to men from twenty to forty years of age. In females, the same rules hold good in general, as far as regards the upper boundary; but the lower margin is wont to project somewhat farther beyond the edge of the ribs than in men, owing to the smaller size of the female thorax.

This remark is still more applicable to children during the first year of life, because in them the organ, and especially the left lobe, is relatively larger, and because the thorax does not undergo great development until the period of puberty.

Percussion is of much service for the determination of the relative sizes and situations of the liver at the sick bedside, and its results may be corroborated in some points by palpation, and still more rarely, by auscultation. In order to yield accurate results, percussion demands certain measures of caution, and some amount of technical experience, without which one may easily fall into error. The stomach and intestines must not be loaded with solid matter, otherwise it will always be impossible to make out the lower margin of the liver. Hence observations immediately after a meal are to be avoided; obstinate constipation must first be removed by means of purging; and accumulations of gas must be got rid of. Boundaries which have been determined during a tympanitic distention of the gastro-intestinal canal must be tested subsequently to the dissipation of the tympanitis; apparent contractions of the liver are often upon this restored to their original size.

For percussion it is best to employ a pleximeter divided into lines or centimètres, and a hammer. In order to ascertain the upper boundary, we ought to percuss from the third intercostal space downwards, until we arrive at a place where the sound is dull. Here the liver is in immediate contact with the wall of the chest; above this lies the convex portion which is covered by a layer of lung gradually increasing in thickness as we proceed upwards. The breadth of this portion, the upper edge of which represents the true upper boundary of the liver, varies from two to five centimètres (four-fifths to two English inches) but usually amounts to three (one and one-fifth of an inch). In a case where precise results are desirable, we must map out the gland all round the right half of the chest, as far as the vertebral column, and indicate its outline with lunar caustic. In ordinary cases, it is sufficient to determine its boundary points in the course of the *linea mammalis* and *linea axillaris*, as also close to the vertebral column, and in the median line in front: in the last-mentioned situation, this is best done by drawing a line from the point of contact of the right side of the cardiac dulness with the upper boundary of the liver to the apex of the heart.

In order to define the lower border of the liver, we percuss downwards until we can distinguish the tympanitic sound of the stomach or intestine. By a percussion stroke of moderate firmness and elasticity this margin is usually made too high,* because the intestinal sound is transmitted through the liver, in cases where the margin of this organ is sharply attenuated, or where the underlying parts contain much gas. To define it with any amount of certainty, we must percuss very gently; and for this purpose, the muffled mode of percussion recommended by Piorry, by making the two or three first fingers strike simultaneously, is of the greatest service. Fine distinctions of sound are in this way more easily recognised.

A mistake of an opposite character not unfrequently happens, owing to the abdominal walls being rendered very tense in consequence of great pain in the epigastric region. Here, from the great muscular tension, the dulness corresponding to the situation of the liver is extended, an illusion which is increased still more by palpation. The inflammations and enlargements (*Anschoppungen*) of the left lobe of the liver, so frequently believed to exist, depend for the most part upon this condition, which in practice is not sufficiently attended to.

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* The situation of this margin can be made out with greatest certainty in cases in which the lower margin can also be felt on palpation; the latter mode of observation almost always indicates the lower margin as somewhat lower down than percussion, unless this is made with great care.

In cases where it appears necessary, we may mark out the lower border in its whole extent, from the extremity of the left lobe nearly as far as the spinal column; but in most cases it is sufficient to determine it in lines corresponding to the nipple, axilla, and sternum. By ascertaining the upper and lower borders at these three situations, we learn the measurements of a third part of the organ, from which we may draw conclusions as to its entire volume.

These measurements, however, in healthy individuals, are liable to variations of considerable extent, the limits of which must be determined, before we can turn the clinical observations to any practical account. A large number of measurements appears to be necessary, in order to become acquainted with the differences arising from age, stature, sex, and other physiological conditions, and in order to ascertain the limits which cannot be transgressed without affording a certain proof of a pathological alteration in the volume of the liver.

TABLE I.

VARIATIONS IN MEASUREMENTS OF LIVER ACCORDING TO AGE AND STATURE.

MALES.*

	Age.	Stature. Centim.	Axillary Line.	Mammary Line.	Sternal Line.		Age.	Stature. Centim.	Axillary Line.	Mammary Line.	Sternal Line.
1.	10 mon.	67	4	3½	½	29.	22 yrs.	160	8	9	6
2.	10 „	69	5	3	2	30.	23 „	153	8	10	8
3.	5 yrs.	103	8½	7	3	31.	24 „	170	9	9	7
4.	6 „	99	6	6	4	32.	24 „	154	9	9	5
5.	7 „	98	8½	7	3	33.	24 „	154	10	10	6
6.	8 „	115	7	7	4	34.	25 „	150	10	10	6
7.	9 „	102	7	6½	4	35.	25 „	151	6	9	5
8.	11 „	125	7	6	3	36.	25 „	158	9	10	6
9.	13 „	125	5	6½	3	37.	26 „	138	12	12	7
10.	14 „	124	7	6	6	38.	27 „	146	8	6	3
11.	15 „	144	7	8	6	39.	27 „	160	10	9	5
12.	16 „	150	7½	10	6	40.	27 „	160	11	10	8
13.	17 „	142	7½	9	5	41.	28 „	160	11	12	8
14.	17 „	146	10	9	5	42.	29 „	150	9	8	6
15.	17 „	144	10	10	7	43.	29 „	150	11	11	7
16.	17 „	162	10	8	6	44.	30 „	170	10	10	9
17.	17 „	149	8	7	5	45.	32 „	160	10	12	6
18.	17 „	154	8	9	7	46.	32 „	154	11	11	6
19.	17 „	157	8	6	7	47.	34 „	150	10	11	3
20.	18 „	151	8	7	4	48.	34 „	156	9	10	6
21.	18 „	150	10	9	7	49.	34 „	152	10	10	4
22.	19 „	156	10	10	6	50.	37 „	160	8	11	5
23.	20 „	155	12	11	7	51.	39 „	168	12	10	4
24.	20 „	168	9	9	7	52.	42 „	171	11	11	8
25.	20 „	153	9	11	6	53.	44 „	150	10	9	4
26.	21 „	155	9	10	6	54.	45 „	166	8	9	6
27.	21 „	155	8	9	5	55.	46 „	160	9	9	7
28.	22 „	164	12	12	5	56.	55 „	155	10	10	4

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* The measurements in this and the following tables are given in centimètres. There are about 2½ centimètres in an English inch, or more correctly, 1 centimètre = .3937 English inch.—TRANSL.

TABLE II.

VARIATIONS IN MEASUREMENTS OF LIVER ACCORDING TO AGE AND STATURE.

FEMALES.

	Age.	Stature.	Axillary Line.	Mammary Line.	Sternal Line.		Age.	Stature.	Axillary Line.	Mammary Line.	Sternal Line.
		Centim.						Centim.			
1.	1½ yrs.	68	4	4	2½	30.	30 yrs.	136	9½	9	6
2.	1½ „	67	4	5	4½	31.	30 „	138	10	8	3
3.	2½ „	80	2½	1½	2	32.	30 „	139	10	9	5
4.	3 „	90	5½	4½	4½	33.	30 „	132	10	10	7
5.	3½ „	76½	4	4	5	34.	30 „	150	10	8	9
6.	4 „	77	2¾	2½	2½	35.	30 „	150	11	10	7
7.	5 „	91	4	2	1	36.	33 „	146	9	9	4
8.	5 „	87	7	7	3	37.	34 „	156	10	11	7
9.	6 „	90	5½	5	4½	38.	35 „	147	10	9	8
10.	11 „	124	10	8	3	39.	36 „	147	8	8	7
11.	12 „	127	7	6	4½	40.	38 „	148	11	9	6
12.	14 „	127	8	9	4	41.	40 „	142	13	11	6
13.	15 „	131	6	7	6	42.	42 „	152	8	9	5
14.	17 „	150	9	9	6	43.	42 „	142	13	12	7
15.	18 „	139	10	10	5	44.	42 „	142	9	8	4
16.	18 „	154	6	8	7	45.	43 „	152	13	8	6
17.	19 „	136	8	9	7	46.	47 „	150	8	9	5
18.	20 „	136	9	8	3	47.	48 „	150	9	8	7
19.	21 „	142	6	7	4	48.	49 „	136	9	8	7
20.	21 „	138	7	8	5	49.	50 „	140	9	8	8
21.	22 „	152	8	9	4½	50.	52 „	155	11	11	6
22.	23 „	146	8	9	5	51.	53 „	144	9	10	8
23.	23 „	158	9	10	5	52.	59 „	148	10	10	7
24.	24 „	147	9	8	5	53.	61 „	146	7	7	6
25.	24 „	152	8	11	6	54.	69 „	137	10	12	6
26.	26 „	151	8	7	6	55.	76 „	146	9	7	4
27.	27 „	150	6	8	5	56.	79 „	144	8	7	7
28.	27 „	152	9	8	6	57.	80 „	154	10	8	5
29.	27 „	142	9	9	7	58.	80 „	150	10	7	4½

TABLE III.

AVERAGE MEASUREMENTS ACCORDING TO STATURE.

Stature.	Axillary Line.	Mammary Line.	Sternal Line.	No. of Individuals.
From 67 to 100 centimetres -	5.09	4.23	2.71	13
„ 100 „ 150 „	8.88	8.55	5.48	58
„ 150 „ 160 „	9.17	9.54	5.88	35
„ 160 „ 170 „	10.00	9.56	6.28	7
„ 170 „ 180 „	11.00	11.00	8.00	1

TABLE IV.—AVERAGE MEASUREMENTS ACCORDING TO AGE.

Age.	Axillary Line.	Mammary Line.	Sternal Line.	No. of Individuals.	Age.	Axillary Line.	Mammary Line.	Sternal Line.	No. of Individuals.
Under 2 years - -	4.25	3.85	2.37	4	From 15 to 20 years	8.89	8.89	5.90	19
From 2 to 6 years -	5.8	4.38	3.72	9	„ 20 „ 40 „ -	9.36	9.5	5.82	49
„ 6 „ 10 „ -	7.55	6.83	3.33	3	„ 40 „ 60 „ -	9.75	9.31	6.18	16
„ 10 „ 15 „ -	7.12	7.6	4.44	8	„ 60 „ 80 „ -	9.0	8.0	5.41	6

TABLE V.—COMPARISON OF MEASUREMENTS IN EITHER SEX.

	Axillary Line.			Mammary Line.			Sternal Line.			No. of Individuals.		
	Males.	Females.	Average.	Males.	Females.	Average.	Males.	Females.	Average.	Males.	Females.	Total.
A. According to Stature.												
From 67 to 100 cent. -	5.87	4.36	5.09	4.87	3.94	4.23	2.37	3.28	2.71	4	9	13
„ 100 „ 150 „ -	8.57	9.04	8.88	8.3	8.64	8.55	5.25	5.74	5.48	20	38	58
„ 150 „ 160 „ -	9.2	9.09	9.17	9.76	9.1	9.54	5.96	5.77	5.88	24	11	35
„ 160 „ 170 „ -	10.0	—	10.00	9.56	—	9.56	6.28	—	6.28	7	—	7
„ 170 „ 180 „ -	11.0	—	11.00	11.0	—	11.00	8.0	—	8.00	1	—	1
B. According to Age.												
Under 2 years. - - - -	4.5	4.45	4.25	3.25	4.55	3.85	1.25	3.49	2.37	2	2	4
From 2 to 6 years. - -	7.25	4.35	5.8	6.5	2.38	4.38	3.5	3.94	3.72	2	7	9
„ 6 „ 10 „ - -	7.55	—	7.55	6.83	—	6.83	3.33	—	3.33	3	—	3
„ 10 „ 15 „ - -	6.5	7.74	7.12	6.62	8.58	7.6	4.5	4.38	4.44	4	4	8
„ 15 „ 20 „ - -	9.07	8.71	8.89	8.92	8.86	8.89	6.07	5.73	5.90	14	5	19
„ 20 „ 40 „ - -	9.61	9.11	9.36	10.0	9.0	9.5	5.85	5.79	5.82	26	23	49
„ 40 „ 60 „ - -	9.6	9.9	9.75	9.6	9.02	9.31	5.8	6.56	6.18	5	11	16
„ 60 „ 80 „ - -	—	9.0	9.0	—	8.0	8.0	—	5.41	5.41	—	6	6

Apart from the rough outlines of the liver which are obtained by means of percussion, palpation of the organ, so far as this is practicable, is of service for determining its form. In practising palpation, everything must be avoided which can induce tension of the abdominal muscles, and thus interfere with the free penetration of the hand. It is most advantageously performed when the patient is in a recumbent posture, and then the abdominal muscles are most easily relaxed by extreme flexion of the thigh, and by raising the upper part of the body by means of pillows placed underneath. The hand, which must of necessity have been previously warmed, should be laid cautiously upon the abdominal wall, and a gradually increasing pressure inwards should be made with the points of the fingers in a rotating manner; by the hasty application of a cold hand, muscular contraction is excited to such a degree, that deep penetration is impracticable. Several attempts are not unfrequently required before an anxious patient is so far composed as not to render the examination unnecessarily difficult through muscular contraction. Many moveable parts, such as a distended gall-bladder, are felt on light application of the hand more distinctly than on firm pressure, by means of which they are displaced and escape from under the fingers; there is no objection, however, to this last mode of examination in the case of solid tumours on the under surface of the liver, although, in order to be successful, palpation must always be practised not abruptly, but with a gradually increasing pressure. The liver is most accessible along the outer margin of the rectus; as far as this muscle extends, its tension causes the organ to be felt with difficulty. Of course the *linea transversa* must not be mistaken for the margin of the liver.

When the margin of the liver has been felt with the points of the fingers, we may, by pressing above and below this margin, ascertain its thickness, and whether it has its normal sharpness or roundness; we may also, in this way, observe where the rounded margin, characteristic of the right lobe in its normal condition, commences, whether the upper surface is smooth or rough, and whether there exist large, painful nodules, or painless processes of the lobe itself, surrounded by deep fissures. In this way, too, we may obtain a knowledge of the consistence of the organ, whether it is hard or doughy, and also of the motility of tumours, which, in the case of the lobes separated by tight-lacing, is not unfrequently so great, that we can turn the tumour and lay it upon the upper surface of the organ. In determining the sizes of tuberosities and excrescences of the liver, we must, of course, before we form an opinion as to their real extent, deduct the thickness of the abdominal walls, one or twofold, according to the mode of examination. The margin of the liver should be traced out, so far as this is practicable, to the right and left, and care should be taken to make out the position of the fissure of the liver, and of the round ligament, inasmuch as a knowledge of their situation is of great service in the diagnosis of displacements of the organ.

In many cases, the situation of the liver is so high, that every attempt at palpation is fruitless; in others, the left lobe only can be felt in the epigastrium, and then the examination must be made with peculiar caution, in consequence of the difficulty occasioned by the rectus muscle.

Inspection of the hepatic region rarely furnishes data for diagnosis, which may not be obtained more accurately and completely by means of percussion and palpation. Visible bulgings of the right hypochondrium, whether smooth or nodulated, not unfrequently result from lardaceous (*speckig*) infiltration, from carcinoma, or from echinococci of the liver; and they may even attain such a prominence, that the boundaries of the organ may be recognised through the abdominal parietes (see Fig. 14); these rare cases, however, only raise new difficulties in diagnosis. In most cases, inspection fails to afford us any assistance, yet on two points, it will be of great service, viz., when there is enlargement of the veins of the abdominal walls, and in reference to the situation of the umbilicus.

In great enlargements and in displacements of the liver, the situation of the umbilicus is carried downwards, so that it approaches nearer to the symphysis of the pubes, whereas in tumours which arise from the bottom of the abdomen, and fill up the whole of the abdominal cavity, it is pushed upwards towards the sternum. These changes, upon which Ballard* appears to lay some stress, must only be made use of with great caution, because the distances of the umbilicus from the base of the ensiform cartilage, and from the pubes, are susceptible of remarkable differences, even under normal circumstances, as shown by measurements which have been made in these localities.

Enlargements of the branches of the epigastric veins may exist in a moderate degree, when the abdominal walls are much distended and tense; where, however, they attain a considerable degree without these last-mentioned conditions, they indicate an obstruction to the circulation of the blood in the vena cava inferior, or in the vena porta. In most cases it is not difficult to determine which of these two vessels is at fault, for when the obstruction has its seat in the vena cava, the venous roots of this trunk are seen to participate in the enlargement from the beginning, whereas they remain for a long time exempt when the portal vein is contracted or closed up. Subsequently, under the heads of cirrhosis of the liver and of diseases of the portal vein, we shall return to this subject, and shall also speak of the enlargement of the veins around the umbilicus, described as the *caput medusæ.*

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* The physical Diagnosis of Diseases of the Abdomen. London. 1852.

Auscultation is of little use for the objects under consideration; it may be of service by assisting in the determination of the diaphragmatic boundary of the liver. As a result of the compression of the lower lobe of the right lung by enlargement or elevation of the liver, there may be heard, according to Walshe, along the line of junction of the two organs, towards the end of a deep inspiration, a dry crepitation, or "hepatic compression rhonchus." In a few cases of this nature, I have heard consonating respiratory murmurs, although, as was proved by *post-mortem* examination, no infiltration of the lungs existed.

B. *The Application to Diagnosis of the Results of Observation.*

This is a less simple matter than might at first sight appear; manifold difficulties, which may lead to mistakes, meet us at every step.

The surface of the liver, which is in contact with the wall of the chest and abdomen, is the part of the organ upon the size of which we must depend for information. The extent of this surface, however, is very variable, even when there is no alteration in the volume of the liver, inasmuch as the position of the organ is liable to change. Extension in a downward direction, such as results from the pressure of tight clothing, from deformity of the lower part of the chest, and softening of the hepatic parenchyma, brings a larger portion of the surface into contact with the abdominal wall, and so produces an apparent enlargement; whilst, on the other hand, elevation of the organ, and the change in position produced by distention of the lower part of the abdomen, bring the anterior sharp margin of the organ in opposition to the abdominal wall, and thus give rise to an apparent diminution. These conditions, as well as congenital and acquired malformations of the liver, constitute our first difficulties in diagnosis; others arise from displacement of the entire organ, and from the obstacles which are experienced in determining its boundaries, owing to pathological changes in the adjoining parts.

1. *Congenital and acquired Malformations of the Liver.*

From its first formation the liver is liable to numerous abnormalities in form, which may easily lead to mistakes in the examination of the organ at the bedside of a patient. In many cases it appears almost quadrangular (Fig. 1.); in others, it is furnished with abnormal fissures and rounded, so that its left margin scarcely reaches to the median line (Fig. 2.); whilst in other cases, the left lobe is prolonged in the form of a tongue, and stretches far into the left hypochondrium (Fig. 3.). When this is the case, the liver sometimes overlaps the spleen, and contracts firm adhesions with the surface of this organ, so that a definition of the boundaries of each is impossible. (Fig. 4.)

FIG. 1.

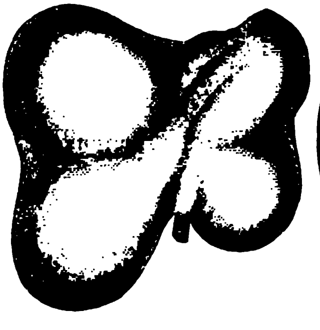


FIG. 2.

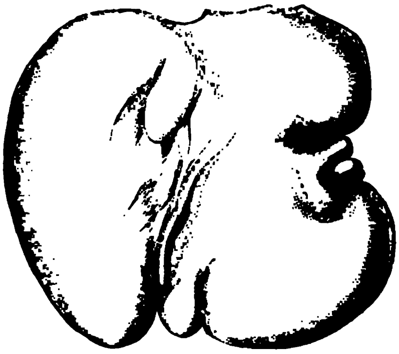


FIG. 1. Liver of an abnormal, quadrangular form: upper surface.
FIG. 2. The lower surface of a liver presenting an abnormally rounded outline, so that when *in situ*, the left lobe scarcely reached to the median line. The right margin is seen to be abnormally fissured.

The size and form of the left lobe are, under normal conditions, so very variable, that the determination of its extent must only be employed for clinical purposes with great caution.

FIG. 3.

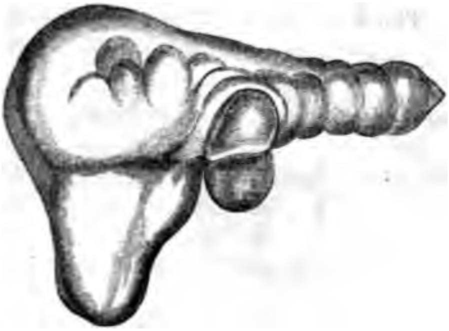


FIG. 4.

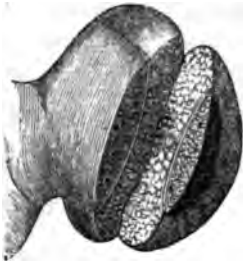


FIG. 3. Liver, with an abnormally prolonged left lobe, and a tight-lace depression.

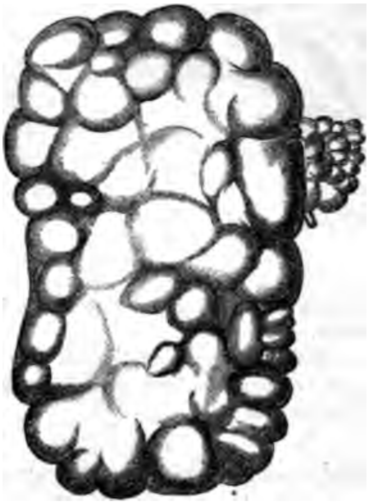
FIG. 4. Adhesion of the extremity of the left lobe of the liver to the spleen; a section has been made through the line of junction of the two organs.

To these congenital malformations, a knowledge of which can only be acquired by frequent *post-mortem* examinations, are to be added, acquired malformations of a still more variable character, partly resulting from disease of the hepatic tissue, and partly from compression of the organ from without, by deformities in the thorax, &c. Independently of tumours, carcinoma, echinococci, and abscesses, which may extend in any direction, the diseases which especially affect the form of the liver are those chronic morbid processes, which terminate in cirrhotic degeneration of the gland, and which usually, though by no means invariably, occasion an undue contraction of the left lobe, and a retraction of this towards the right side. The annexed figure, reduced to one-fourth of the natural size, shows what a remarkable disproportion of the two lobes may result from such a state of matters. (Fig. 5.)

Here, also, we must include the lobulation of the organ which arises from obstruction of several branches of the portal vein, and from partial inflammations and cicatrization of the hepatic parenchyma, as also those rare cases of congenital lobulation, of a nature similar to what is observed in the case of the kidneys.

Much more frequently than these deformities, we meet with, especially in females, abnormalties in the form of the liver produced by tight-lacing of articles of clothing and lateral distortion of the thorax. Provided there is narrowing of that part of the walls of the chest which encloses the liver, we have, as a result of lateral distortion, either displacement of the organ or an alteration of its form. Sometimes the liver is rolled up into a rounded conical mass, but more frequently it is more or less deeply notched, by the turning inwards of the margin of the ribs. From its daily occurrence, the so-called tight-lace liver (*Schnürleber*) has an importance in diagnosis greater than is otherwise its due.

FIG. 5.

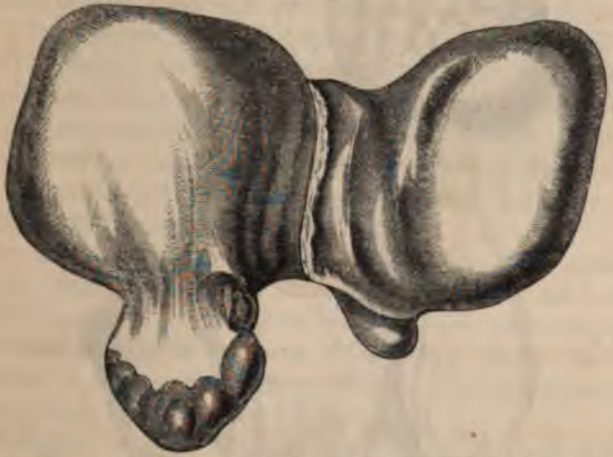


Cirrhosis of the liver, with remarkable atrophy of the left lobe.

According to the changes in the fashion of dress, tight-lacing in producing a thin waist, affects sometimes a higher and sometimes a lower part of the thorax, and more rarely the hypochondrium beneath the ribs. Of all the organs, the liver suffers the most from this pressure; not only is its form altered, but in most cases, if we examine it at a late period, its situation and position also. By the narrowing of the base of the thorax, the organ is first compressed from side to side, and as a consequence of this, especially when its transverse diameter is considerable, there not unfrequently, is produced a series of folds, which feel like slightly prominent nodules (Fig. 3).

At the same time, as a consequence of the circular contraction, a part of the right, and usually of the left lobe also, becomes separated by a depression, the situation of this depression being sometimes higher and sometimes lower, according to the locality of the lacing. The furrow thus formed often penetrates deeply into the parenchyma, till there remains nothing more than a loose ligamentous connection, which allows a free motion of the separated portion (Fig. 6).

FIG. 6.



Abscission of the right lobe of the liver, with thickening of the capsule.

The serous covering of this portion always appears thickened, and of an opaque white, and the biliary ducts may be seen through the peritoneal coat, enlarged and full of a brownish mucus, the evacuation of which is prevented by the constriction (Fig. 7). The veins are invariably enlarged. The margins of the detached portion are rounded and nodulated (Figs. 6 and 8), and its tissue feels firmer, and exhibits a finely-granular appearance, similar to that which we find throughout the entire organ, when there is an obstruction to the circulation of the blood in consequence of disease of the heart.

In this way there is produced a moveable nodulated tumour in the situation of the right lobe of the liver, which feels hard and

FIG. 7.

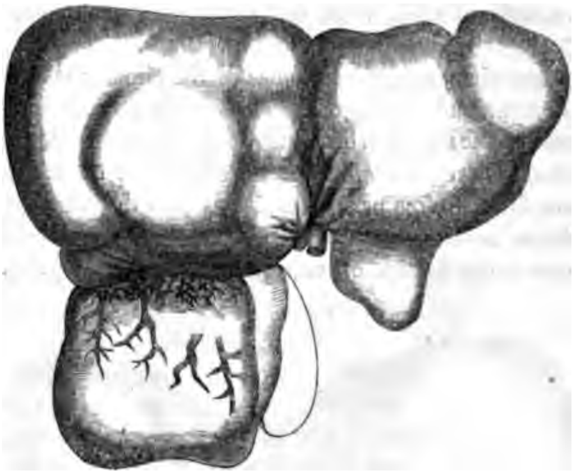


FIG 8.

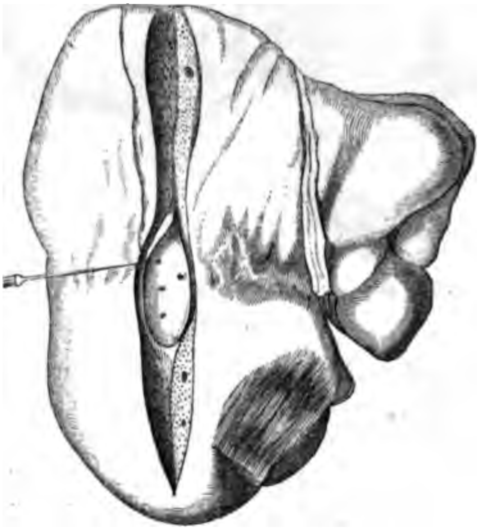


FIG. 7. Abscission of both the right and left lobe of the liver, with enlargement of the bile ducts and of the veins below the tight-lace fissure.

FIG. 8. Abscission of the right and left lobes of the liver: a section is made showing enlarged vessels in the tight-lace constriction.

nodulated, and may hence be easily mistaken for a new growth. A smaller tumour which may be grasped in the fingers may also be developed upon the left lobe. The possibility of an error in diagnosis, arising under such circumstances, is still farther increased by the fact, that the situation and position of the entire organ are almost always altered in various manners at the same time.

2. *Abnormal Positions of the Liver.*

By abnormal positions we understand deviations from the normal direction of that axis of the liver, which extends from the anterior sharp margin to the middle of the posterior rounded border. This may incline very much downwards, in which case, a large portion of the convex surface of the organ is opposed to the abdominal parietes, and the liver appears larger; it may, on the other hand, be elevated to such a degree that the sharp anterior margin alone remains opposed to the abdominal wall, and thus the extent of surface yielding a dull percussion sound is reduced to a minimum. The *ligamentum pensorium hepatis* restricts the movement of the liver only in cases of enlargement; as Hyrte justly remarks, this is not to be regarded as the true supporter of the liver.

There are various causes which give rise to these abnormal positions. Sometimes they are the result of mechanical force acting upon the organ from above, or from below; at other times they result from disease, followed by softening of the hepatic tissue. In many cases, there is simply change in position of the organ; but more generally, along with this, there is dislocation.

One frequent cause of abnormal positions of the liver is tight-lacing, the operation of which varies according to the part of the gland which is subjected to compression. When the middle or lower portion of the liver suffers from the circular compression of stays, or more commonly of the petticoat strings, the axis of the organ falls downwards, and the edge of the right lobe protrudes far below the margin of the ribs, so that sometimes it may be felt in the region of the cæcum close to the crest of the ilium.* The removal of the hepatic parenchyma along the groove produced by the tight-lacing, contributes also to the widening of this groove. When the groove is deep, coils of intestine not unfrequently lie in it, and a round solid tumour may be felt in the right iliac region, separated to all appearances from

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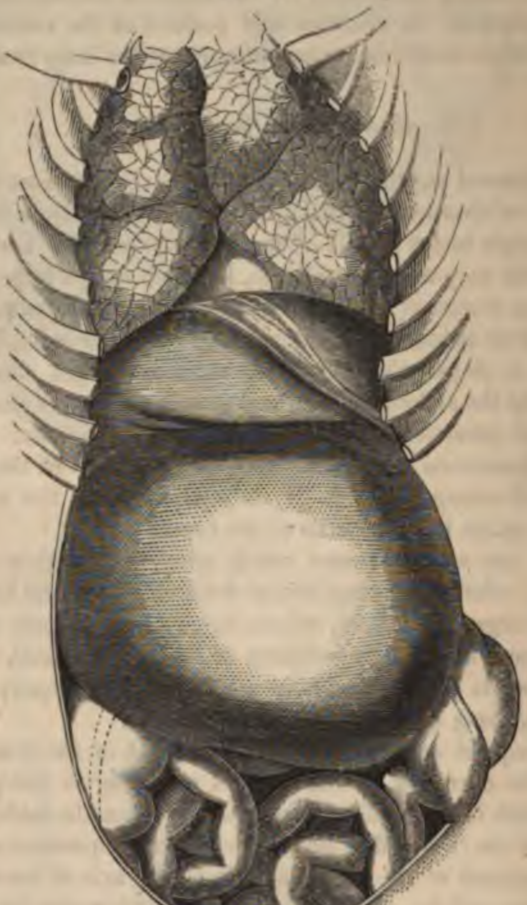
* Cruveilhier(Livr. 40, pl. I. p. 5) asks the question, how a liver which has been altered in position through the operation of a tight-laced chest, may be distinguished from that which results from inflammatory enlargement; and assumes that the former may be diagnosed from its sharp margin. This is a mistake. The right lobe, which is the only part that comes under observation, has always, as the result of tight-lacing, a nodulated rounded margin.

FIG. 9.



Dislocation of the liver towards the median line from tight-lacing; a depressed furrow on the upper part of the right lobe; apparent enlargement of the organ.

the liver by a space, yielding on percussion a clear tympanitic sound. If the left lobe is small, it may remain quite unaffected by the tight-lacing; there may, however, be often found upon its margin a moveable rounded detached portion, which can be pushed backwards or forwards. The upper boundary of the organ is either quite unaffected, or may be carried upwards towards the thorax, owing to the application of a constricting force over the upper portion.

When, on the other hand, the upper third of the liver, where its substance is of considerable thickness, is compressed, the axis is also carried very much downwards, and in most cases the entire organ is at the same time dragged over towards the middle line. The round ligament may then be found opposite the eighth or ninth left costal cartilage, and the middle of the right lobe may correspond to the linea alba, or may even lie to the left of this, whilst the left lobe penetrates deeply into the left hypochondrium (Fig. 9). Upon examination, a liver of about the normal size is then, to all appearances, remarkably enlarged, from its filling up the entire upper-half of the abdominal cavity: even upon *post-mortem* examination, this may be the first impression, until we are convinced of the contrary, by measuring and weighing the organ.

When, as at the present day rarely happens, the lacing is made below the liver, or close to its lower edge, the organ is pushed up towards the cavity of the chest, and its upper boundary is elevated one intercostal space or more.

The confusion of the alterations in the situation of the liver just alluded to, with real enlargements and tumours of the organ, is, after a little practice, easily avoided, inasmuch as, by means of careful palpation, we may detect the depression produced by the lacing; the marks of the pressure are also often visible upon the skin. It is not, however, to be supposed, that these tight-lacings should always have a remarkable effect upon the arch of the ribs; as soon as the tight articles of clothing are loosened, the depression disappears in a great measure, and is then easily overlooked, unless the pressure exerted has been very great and prolonged. We shall return at greater length hereafter to the diagnosis between the condition of the liver resulting from tight-lacing, and several real diseases of the organ.

Under certain circumstances, lateral distortion produces the same effects as stays: tumours also, appearing between the liver and diaphragm (unless they lie very far back), and likewise circumscribed peritoneal exudations, &c., compress the organ in a downward direction.

The left lobe of the liver may, when perfectly healthy, undergo slight alterations in position, which may be a source of error, owing to changes in the condition of the stomach. When this organ is empty, the left lobe is much depressed, whilst it is elevated when the stomach is full.

A change in the position of the entire liver in an opposite direction to that above noticed is observed, when large quantities of gas accumulate in the stomach and intestinal canal, or when from ascites, &c., the parts of the intestine which contain air are pushed upwards against the diaphragm. The anterior margin of the liver is then elevated, so that the surface which is in contact with the abdominal parietes is always reduced in extent. Not unfrequently, the anterior sharp edge is the only part which remains opposed to the abdominal wall. When this is the case, the dull space on percussion diminishes more and more, until it becomes difficult to detect the liver at all, at the place where the pulmonary sound passes into that of the intestine. In the mammary and axillary lines, it is often quite impossible to do this, because in these situations the margin is surmounted by the distended intestines, but in the axillary line, such an occurrence is exceptional.

In this state of matters the liver appears considerably diminished, although in reality, its volume is in no way altered. Hence it is a rule which should never be broken, that diminutions of the hepatic dulness, observed in the course of typanites or ascites, and accompanied by elevation of portions of the bowel containing gas, should only be made use of in diagnosis with great reserve. Oft-repeated observations are here indispensable. After the removal of the gases, the results of percussion are frequently entirely different from what they were before. When there has been protracted constipation, the bowels should in the first place, be cleared out by means of purgatives.

Structural diseases of the hepatic parenchyma give rise to abnormal positions of the organ only when they are accompanied by softening. This is the case in fatty degeneration and in acute atrophy. The effects of these diseases vary according to the degree of softening. When this is moderate, as may happen in fatty liver, the axis falls downwards, and the gland protrudes farther below the margin of the ribs. Hence a fatty liver usually appears upon percussion to be larger than it really is. When the softening is considerable, as in acute atrophy (in which the gland is flexible like a piece of cloth), the liver folds upon itself, and collapses towards the vertebral column, and the space corresponding to it in front is filled up by intestines containing gas. The hepatic dulness then dis-

FIG. 10.

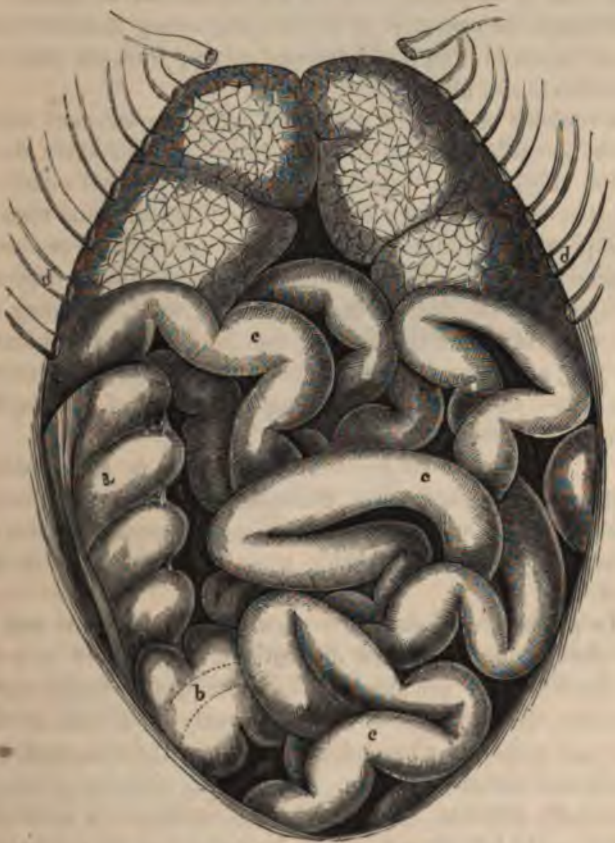


FIG. 10, represents the situation of the parts in a case of acute atrophy of the liver. The organ has become folded upon itself; collapsed towards the spine, the space corresponding to it being occupied by folds of intestine: *a*, colon ascendens; *b*, sigmoid flexure; *c*, jejunum; *d*, sixth rib. The details of the case will be found under Observation XV.

appears entirely in front, whilst posteriorly it may still be detected. Fig. 10 represents the relative position of the different parts in a case of acute atrophy. *a.* Ascending colon. *b.* Sigmoid flexure. *c.* Small intestine. *d.* Sixth rib.

3. *Abnormal Situations. Dislocations of the Liver.*

The liver is frequently entirely dislodged from its normal situation, through the operation of forces compressing it either from the chest, or upwards from the abdominal cavity; from the same causes its position is also altered.

The contents of the thoracic cavity exercise a constant influence over the situation of the liver, inasmuch as with every deep inspiration the organ is depressed from one to one and a-half centimètre (two-fifths to three-fifths of an English inch), and rises again upon expiration. Protracted dislocations downwards are produced by all diseases of the thoracic organs, which permanently depress the diaphragm; but the altered situation varies greatly, according as the pressure operates from the right or the left side of the thorax, and partly also, according to the nature of the disease which occasions this pressure, whether it acts more upon one part of the diaphragm than upon another.

a. In pulmonary emphysema, when this is so considerable that the lungs exceed their normal dimensions, the liver falls downwards from one to one and a-half intercostal space. The displacement is most remarkable when the right lung is chiefly affected.

b. In pleurisy, with abundant purulent effusion,* and still more so in pneumothorax, marked dislocations of the liver in a downward direction are produced. Not unfrequently under such circumstances, the diaphragm is turned convexly downwards, so that its lowest part projects far beyond the margin of the ribs, pushing the liver before it into the abdomen. The resulting situation of this gland is essentially different, according as the exudation is contained in the right or the left pleural cavity. In pleurisy of the right side, the diaphragm becomes arched convexly downwards, and depresses the right lobe of the liver, so that its anterior border reaches several inches beyond the ribs. In the case represented in the annexed figure, the anterior margin of the right lobe of the liver (*b*) was eight inches below the apex of the ensiform cartilage. (Fig. 11.)

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* Slight fibrinous exudations may exist without any remarkable effects upon the situation of the liver.

FIG. 11.

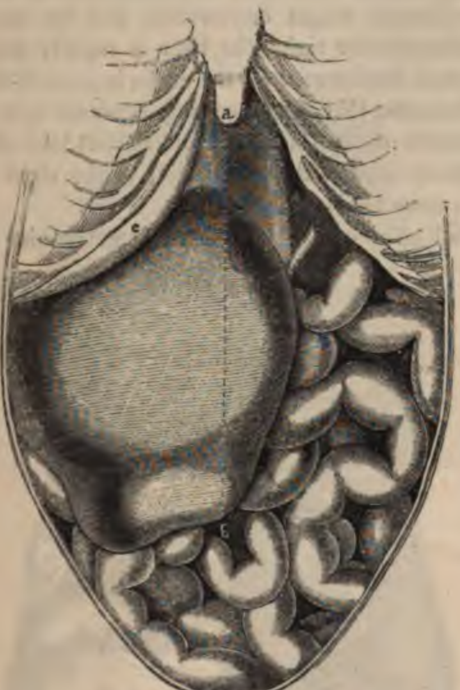

FIG. 11. Dislocation of the liver resulting from exudation into the right pleural cavity: *a*, ensiform cartilage; *b*, lower margin of the right lobe of the liver; *c*, diaphragm arched downwards.

The liver is at the same time pushed towards the left side, and the suspensory ligament is stretched obliquely to the left of the median line. The left lobe is not usually depressed, but on the contrary, owing to the point of suspension of the organ from the suspensory ligament acting as the fulcrum of a lever, it is pushed upwards and to the left, the apex of the heart lying in front of it. This elevation is, however, by no means of universal occurrence; it is completely absent when, as often happens, the left lobe is too thin and soft to overcome the resistance from above, or when the mediastinum is pushed by the pleuritic effusion over towards the left, so that it presses upon the left lobe equally with the right.

In pleuritic effusion on the left side, the change in the situation of the liver is entirely different. It is true that in this case also, when the diaphragm bulges downwards, and the mediastinum is pushed over towards the right, the liver is slightly depressed; but still, the principal displacement of the organ is towards the right, and is of such a character that the suspensory ligament is transported to opposite the eighth or ninth right rib, and the left lobe of the gland is carried completely beyond the middle line to the right side, and at the same time downwards. (Fig. 12.)

c. Pericardial effusions and eccentric hypertrophies of the heart

FIG. 12.

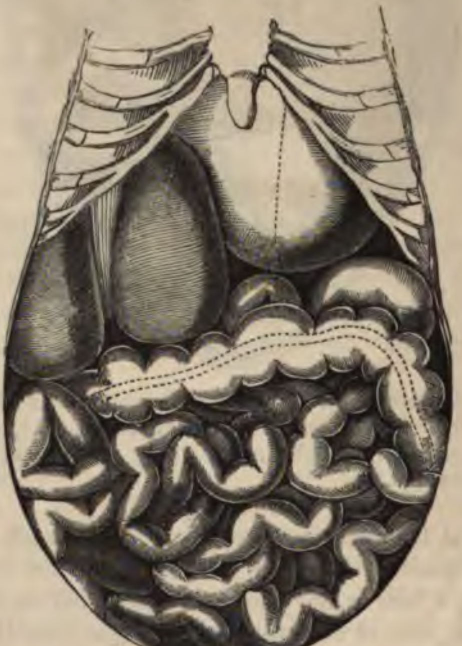

FIG. 12. Dislocation of the liver towards the right side, resulting from effusion into the left pleural cavity. In this case, the diaphragm projected 4½ inches beyond the lower margin of the ribs.

exercise but little influence over the situation of the liver. The former, however, when considerable, may depress the gland to the extent of half or a whole intercostal space, and in this case it is the left lobe which is chiefly affected. (Fig. 13.) The congestive swelling of the liver which is usually present under such circumstances, increases this apparent change of situation, which, therefore, must not be judged of from the degree of bulging of the organ below the margin of the ribs.

FIG. 13.

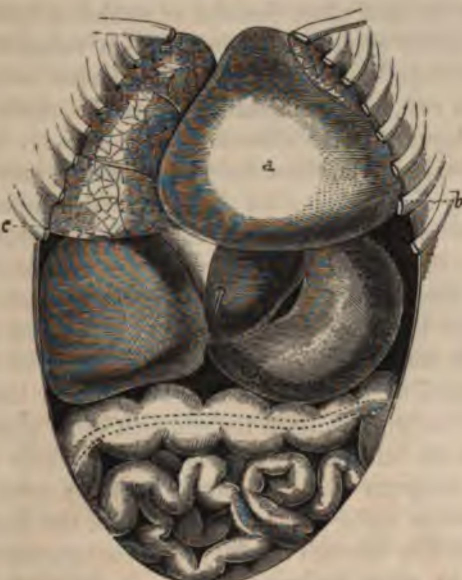

FIG. 13. Dislocation of the liver, especially of the left lobe, downwards, resulting from a remarkable effusion into the pericardium: *a*, pericardium; *b*, sixth rib.

In general, it is not difficult to distinguish between the changes in the situation of the liver just mentioned, and hypertrophies of the organ itself. This is especially an easy matter in emphysema and pneumothorax, in which the low situation of the upper boundary is at once made out by means of percussion. Errors in diagnosis are at the present day quite unjustifiable; such, for instance, as I have met with in one consultation, where it had to be decided, whether the recommendation of the Marienbad waters, by two experienced physicians, was suitable for a case of suspected enlargement of the liver, which turned out to be an example of displacement of this gland, resulting from pneumothorax supervening upon pulmonary tubercle. It is a more difficult matter to come to a decision in certain cases of dislocation of the liver from right pleuritic effusion, and especially in cases where the layer of exudation in its height and situation does not exceed limits to which an enlarged liver might possibly have risen.* Under such circumstances, when, from the history of the case, no clear information can be obtained as to the mode of development of the affection, and when we are unable to distinguish the conical form, characteristic of tumours, growing upwards into the cavity of the chest from the horizontal boundary, which in most cases distinguishes pleuritic effusions, we must be guided chiefly by the mobility of the liver upon deep inspiration. When this remains unchanged, as can in most cases be made out without any difficulty,† by means of palpation and percussion during a full inspiration, we conclude that the diaphragm, and with it the liver, are not depressed by pleuritic effusion, but that an increase in the volume of the organ has altered the situation of its upper and lower margin. On the other hand, we must not infer with certainty the existence of empyema from an absence of the natural mobility of the liver, because quite independently of this, the diaphragm may lose its contractility through the fatty or fibrous degeneration, which this muscle so often undergoes, when it becomes adherent to tumours of the liver. This is very often the case with tumours reaching high up into the thorax.

In addition to the mobility of the liver upon inspiration, it should be noted, whether the cartilaginous margin of the arch of the ribs on the right side appears to be turned outwards, a circumstance of frequent occurrence in enlargement of the liver, but which is not observed in empyema.

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* This may be considerable: tumours of the liver, as, for instance, echinococci, which grow from the upper convex surface of the organ, not unfrequently elevate the diaphragm as high as the third or second rib. In such a case, when there is also pleuritic effusion, the definition of the upper margin of the liver will depend upon the slight resistance of the swelling growing on the thoracic side of the diaphragm, as compared with that on the abdominal side.

† We rely here upon the situation of the lower margin of the liver, inasmuch as, even when the diaphragm is inactive, there is an apparent depression of the upper boundary during a deep inspiration, in which, owing to the altered position of the ribs, and the enlargement of the thoracic cavity, the margin of the lung extends downwards between the liver and the wall of the thorax.

Stokes places great stress in diagnosis upon the depression, which in cases of effusion into the thorax may be made out between the margin of the ribs, and the convex upper surface of the liver. This (as represented in Fig. 11.) is produced in the line of contact of the right half of the diaphragm bulging convexly downwards with the upper surface of the liver, and may be often distinctly felt, and sometimes also seen, during life; but for the purpose of diagnosis, it is for several reasons of little value. In empyema producing a bulging downwards of the diaphragm, where the layer of exudation extends to a considerable height, the diagnosis is in most cases easy, quite independently of the existence of this sign; in the more obscure cases, in which this sign would be of value, it is wanting, because here there is not such a remarkable displacement of the diaphragm as to produce it. Frequently, moreover, even when the depression exists, it cannot be detected, owing to the tension of the abdominal muscles being so great. Lastly, a similar furrow may exist in the case of all nodulated tumours of the liver, such for instance, as in very prominent carcinomatous and hydatid tumours, for by the bulging which they occasion, the liver is removed from the thoracic wall, and thus there is created a space between it and the ribs large enough to admit the finger.

The following case, which is also interesting in other respects, shows that the obliteration and fluctuation of the intercostal spaces, with displacement of the mediastinum are not, as has been assumed, certain proofs of the existence of empyema.

OBSERVATION No. I.

Tertiary Syphilis.—Tumour of the liver, which ascended as high as the second rib, and consisted of echinococci along with lardaceous infiltration.—Bulging and fluctuation of the intercostal spaces.—Displacement of the heart.—Immobility of the tumour upon deep inspiration.—Diagnostic value of exploration by a trocar.

Wilhelmina Köhler, aged 38, who had formerly been repeatedly treated for secondary and tertiary syphilis, was admitted into the clinical wards on January 15th, 1855, with œdema of the feet and ascites. On examination of the abdomen, there was found a remarkable enlargement of the liver and spleen. On the right side there was dulness on percussion, extending from the fourth rib to three centimètres above the umbilicus; on the left side, it appeared impossible to define the boundaries of the spleen. The surface of the liver was somewhat uneven; its margins felt hard and resistant. The spleen extended as far forwards to the anterior extremities of the tenth and eleventh ribs; higher up, its dulness was continuous with that of the liver. Appetite normal; bowels regular; stools of a pale brownish colour; the urine scanty, and of a dirty-yellow colour, but free from albumen and bile-pigment. The colour of the skin was a yellowish-grey; nutrition was but slightly impaired; the menses had ceased for three months. Upon the shin-bones there were syphilitic nodes, which were painful towards night; and old cicatrices could be seen upon the velum-palati. The diagnosis arrived at was syphilitic lardaceous degeneration of the liver (*syphilitische Speckleber*) and spleen. Iodide of iron, along with a diuretic infusion, was prescribed.

The œdema of the lower extremities completely disappeared after some days, as did, also, in a great measure, the ascites; the liver and spleen, however, underwent no alteration in volume, although the preparations of iodine (iodide of iron, and afterwards iodide of potassium) were continued for ten weeks.

During the vacation, which the patient spent in another part of the hospital, her condition underwent an essential change. A circumscribed pleuritic effusion, causing displacement of the liver and of the heart, was reported to have made its appearance in the front of the right side of the chest, without the ordinary subjective phenomena of such a lesion.

At the end of April, when the patient again came under observation in the clinical ward, the following facts were noted:—The right side of the thorax was distended; in front, the respiratory murmur was completely absent from the second rib downwards, and there was dulness on percussion from the same place down to a level with the umbilicus, and in a line with the mamma as far as six centimètres (2⅜ English inches) below this; posteriorly, vesicular breathing was audible as low as the ninth rib. The fissure of the liver lay immediately behind the depression of the umbilicus, and from this point the left lobe extended to below the cartilage of the tenth left rib, where, in the axillary line, it was bounded by the tympanitic stomach-sound. The heart was pushed upwards and to the left, and its pulsation could be seen and felt in the fourth intercostal space, three centimètres (1⅕ of an inch) to the left of the nipple, *g*. On the left side of the chest,

FIG. 14.

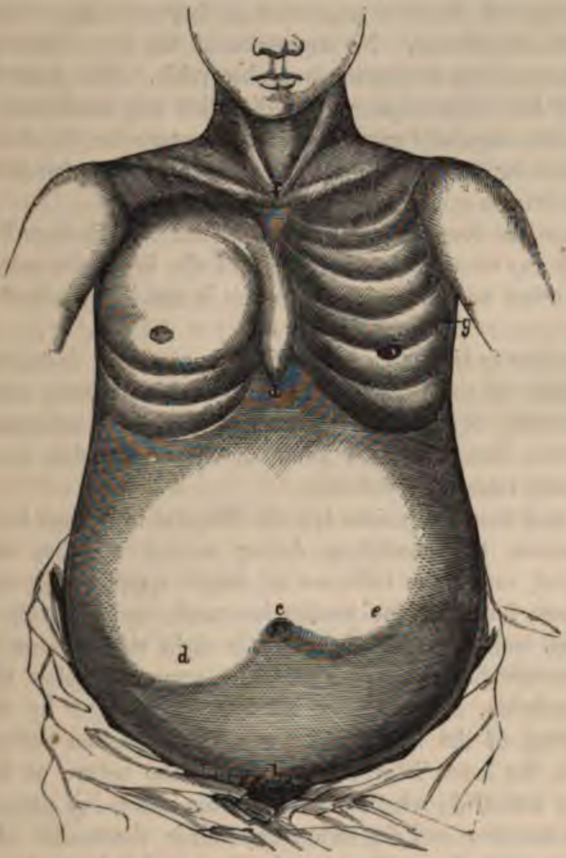

FIG. 14. The appearance presented during life by a patient suffering from echinococci of the liver; *a*, ensiform cartilage; *b*, symphysis pubis; *c*, *d*, *e*, contour of the liver; *g*, situation of the cardiac impulse. The left lobe of the liver has been represented somewhat too large, and extending too low down.

slight dulness on percussion, and bronchial breathing, could be detected at the lower and back part—the remains of a pleuritic effusion, for which the patient had been under treatment on a former occasion, but otherwise, there was nothing abnormal. The surface and margins of the liver presented, as formerly, slight unevenness and a firm consistence. No depression of the lower margin of the organ upon a deep inspiration was observable. The general health appeared but little impaired, the digestion was unaffected, and the bowels were regularly moved, after a spontaneous diarrhœa which had lasted for a fortnight; dyspnœa was only complained of upon moving or upon going up stairs.

The use, for four weeks, of the Karlsbad Mill Springs* did not result in any remarkable diminution of the liver; its margin only became softer and more flabby, so that it could be doubled in upon itself.

From time to time, there appeared symptoms of inflammation of the capsule of the liver (*perihepatitis*), which passed away either spontaneously, or after the application of a warm cataplasm; and there were, also, bleedings from the nose occurring once every month with tolerable regularity.

Now and then the patient left the Hospital to arrange her domestic concerns, her condition, during several months, remaining unchanged, under the influence of simple symptomatic treatment. By degrees, however, the ascites increased, and the veins of the abdominal wall, especially those of the right side, became enlarged to a considerable extent. There also appeared below the right hypochondrium, a fluctuating bulging, surrounded by the solid parenchyma of the liver; and a similar fluctuation could be detected in the lower intercostal spaces. The nature of this fluid appeared doubtful; whether it was serum which lay circumscribed between the liver and the abdominal wall in the meshes of fibrous bands, resulting from oft-repeated attacks of inflammation of the capsule of the liver, or whether it consisted of pus from a pleuritic effusion pressing down the diaphragm, or from an abscess of the liver, or, lastly, whether it was the thin, watery contents of an hydatid cyst. The data furnished by the history of the case, and by direct examination, were insufficient for determining this question. A specimen of the fluid, therefore, was cautiously drawn off on the 8th of June, 1856, by means of a fine exploratory trocar. It consisted of a very yellow, clear water, which deposited scarcely a trace of albumen upon the application of heat or the addition of nitric acid, but which contained leucine, and, so far as might be argued from its reaction with chloride of iron, salts of succinic acid. Thus the fluid must have originated from the cysts of an echinococcus. With the determination of this point, all idea as to the existence of a circumscribed pleuritic effusion on the right side disappeared—an opinion which had become more than probable, chiefly from the information communicated to us, which had been confirmed by the absence of any change in the situation of the liver dependent upon respiration. The displacement of the liver and heart were sufficiently explained by the extension upwards of the hydatid mass.

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* The Mill Spring (*Mühlbrunn*) is one of the sources of the Karlsbad water. The Karlsbad Springs are thermal; their temperature varying from 122° to 197° Fahr. Their principal chemical constituents are carbonic acid, the sulphate and carbonate of soda, chloride of sodium, the carbonates of lime and magnesia, with traces of iron, manganese, iodine, bromine, &c. They are much resorted to for hepatic diseases, and calculous affections.—TRANSL.

On the 16th of July, the patient left the Hospital for some days, and exposed herself injuriously, so as to bring on a fatal termination. By the time of her readmission on the 21st, there had arisen considerable œdema of the feet, and extensive catarrh of the air-passages along with œdema of the lungs.

On the morning of the 23rd, shivering, pulse 130, great dyspnœa, expectoration of frothy fluid mixed with blood, general lividity. Death from asphyxia occurred about noon.

Autopsy.

The diaphragm on the right side reached as high as the second rib. Above this, there could only be seen in front a bluish-grey membrane, destitute of air, and compressing the middle (*b*) and upper lobes of the lung; the lower lobe lay pushed away to the back wall of the thorax, firmly adherent to this and to the diaphragm. In the left side of the thorax, the heart (*e*) was pushed upwards and to the left, and had assumed a transverse position, its apex lying in the third intercostal space. The diaphragm upon this side reached nearly as high as the fourth rib, being pushed upwards by a considerably enlarged spleen, which had applied itself horizontally upon the upper surface of the left lobe of the liver and lay in contact with the blunt upper extremity of the suspensory ligament. The surface of the liver was united, at many places, to the abdominal wall and to the diaphragm by adhesions, partly recent, and partly of old date.

FIG. 15.

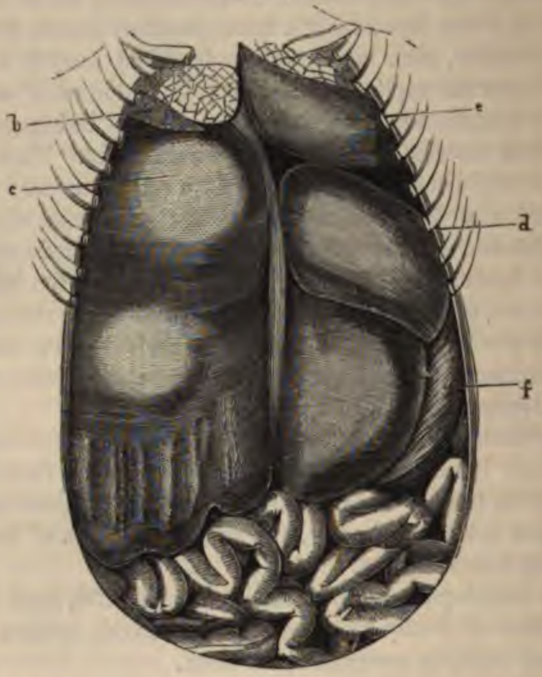

FIG. 15. Arrangement of the viscera from the individual represented in Fig. 14. *b*, middle lobe of right lung; *c*, large hydatid cyst; *d*, spleen pressed upwards by the left lobe of the liver; *e*, pericardium; *f*, stomach.

The left lobe was intimately adherent to the spleen. The muscular tissue of the diaphragm appeared of a pale-yellow colour; upon closer examination, it proved to be in an advanced stage of fatty degeneration. The upper part of the right lobe of the liver was made up of one cyst, the transverse diameter of which was estimated at 9¾ inches (10⅓ English inches). It contained thirteen pounds of a bright-yellow, turbulent fluid, and a single large gelatinous bladder, having its inner surface covered with young echinococci. The fluid had undergone a great change in consequence of inflammation induced by the puncture; it now deposited, upon the application of heat, an abundant quantity of albumen, which had not been present in the specimen drawn off during life. The surface of the liver was uneven, its parenchyma granular, of tenacious (*zähe*) consistence and of a light greyish-brown colour. The entire weight of the liver amounted to 8.68 kilogrammes (19⅛ English pounds avoirdupois); the longitudinal diameter of the right lobe measured 14½ (Paris) inches; the transverse, 8½; the corresponding dimensions of the left lobe were 6 and 4¾ inches, and the thickness of the organ amounted to 7 inches. The spleen was of firm consistence, and of a reddish-brown colour, with a glistening surface on section; it weighed 0.4 kilogramme (14¼ ounces avoirdupois); its length was 6 inches, and its breadth, 4.*

In the cranial cavity, there was nothing abnormal; the mucous membrane of the bronchi was of a bright-yellow colour, and covered with bloody froth; both lungs were very œdematous; at the lower margin of the left lung, the pleura was thickened, and the parenchyma was indurated and traversed by enlarged bronchi.

The right side of the heart was enlarged, but in other respects the organ was normal.

The mucous membrane of the stomach and of the intestinal canal was pale, and that of the latter was covered with greyish-yellow fæces; upon the fundament there was a white cicatrix, extending into the rectum, and measuring 1½ centimètre (⅝ inch).

The urinary and genital organs presented nothing worthy of notice.

Other observations of a similar nature will be given when we come to treat of hydatids of the liver.

d. The displacements of the liver, which are dependent upon abnormal conditions having their seat in the abdominal cavity, take, for the most part, a direction upwards, and are then almost always associated with alterations of the axis of the organ. Everything which fills up the abdominal cavity and narrows the space for the intestines, or which distends these to a great extent, may give rise to such displacements: such, for example, as ascites, pregnancy, ovarian tumours, tumours originating from the omentum, or from the kidneys, and, lastly, tympanites, and accumulations of the fæces in the gastro-intestinal canal. Under such circumstances, displacements of the liver almost always ensue from the inflated coils of intestine rising upwards towards the hypochondrium, and pushing the gland before them towards the thorax, so that its upper border is found behind the fifth, or sometimes even behind the fourth, rib; the anterior sharp margin, which is moveable, is especially elevated, and is often nearly in contact with the thoracic wall, so that in this way the extent of dull percussion-sound is always reduced, and in the median and mammary lines is scarcely distinguishable.

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* See Note, page 18.

It happens much more rarely, that abdominal tumours originating from below, press the liver directly upwards without altering its axis. Ovarian tumours, however, morbid growths in the kidneys, &c., sometimes reach the liver and push the organ upwards, so that we can understand how the direction of its axis should be liable to numerous modifications, according to the more or less lateral operation of the pressure.

It is only in exceptional cases, that pathological changes within the abdominal cavity displace the liver downwards or laterally. Displacement downwards is observed to result from circumscribed peritoneal exudations lying between the diaphragm and the liver, or from hydatids similarly situated; whilst lateral displacements are chiefly produced by great dilatation of the neighbouring portions of intestine, especially of the colon, as a consequence of constriction or compression of more deeply-situated portions, such as the sigmoid flexure, the left curve of the colon and the rectum. I have myself observed great displacement of the liver towards the right concavity of the diaphragm and the ribs of the same side, with consecutive atrophy of the organ, in one individual, in whom, as a consequence of circumscribed peritoneal exudation in the left hypochondrium resulting from a perforating ulcer of the stomach, the descending colon had been compressed, and an accumulation of fæcal matters and gases had taken place in the transverse colon. I have noticed a similar displacement of the liver in the *post-mortem* examination of a man 60 years of age, who died of carcinomatous stricture of the descending colon. At this very date I have under treatment a female with stricture of the rectum from syphilitic cicatrices, in which the colon is almost always distended by gas, or fæculent matter, and pushes the left lobe of the liver across the median line towards the right side.

4. *Difficulties in determining the boundaries of the liver, arising from pathological conditions of the adjoining parts.*

A large proportion of the mistakes in examining the liver, which are made in practice, arise from these causes. Most of them, indeed, may be entirely avoided by a careful examination of the relations of the parts, and with a little practice and experience; but in some cases the illusion is so complete, that it is a difficult matter to escape being deceived. In many such cases, a great point is gained if we can only determine with certainty that the enlargement of the liver is merely apparent, although its volume cannot be positively determined at every part.

The difficulties in determining the boundaries of the liver may proceed from very various parts and organs.

a. From the Abdominal Parietes.

The muscular tissue of the abdomen usually exhibits a remarkable degree of tension at such parts as lie over organs which are the seat of pain. In the hepatic region, such partial muscular contractions are frequently observed as a consequence of catarrh and inflammatory conditions of the pyloric portion of the mucous membrane of the stomach, or of circumscribed peritonitis, perihepatitis, gall-stones, &c. These contractions render the examination difficult, inasmuch as from their hardness and resistance upon palpation, an inexperienced observer may easily mistake them for enlargements of the liver, especially of the left lobe. Such a mistake is avoided by percussion, which does not yield the dull tone, that might have been expected from the degree of resistance on palpation. The percussion-sound is always, however, in such a condition, somewhat shorter and more muffled than natural, in consequence of the violent contraction—a circumstance which must not be allowed to lead us into error.

It is a more difficult matter to avoid mistaking inflammatory infiltrations into the abdominal muscles, for swelling or inflammation of the liver, if, as now and then happens, the disease of the muscular tissue corresponds to the form and situation of this gland. I have met with one case of this nature, in which the resemblance was so striking, that an old and experienced physician had, in order to remove an enlargement of the liver (*Leberanschoppung*), made the patient drink of the Karlsbad Springs for many weeks before he presented himself at the Clinique. In this case, cataplasms after eight days brought an abscess to maturity, and that which the Castle Spring* had been unable to remove, was easily emptied by means of a bistoury. The diagnosis between the two conditions is generally not difficult, because muscular inflammation is rarely limited precisely by the contour of the subjacent liver. Where this happens to be the case, the tightness of the skin over the diseased portion of the parietes, the absence of the other abnormal conditions of the liver, and the insignificance of the general disturbance of the system, enable us to distinguish the true nature of the affection. In addition, a satisfactory confirmation is afforded by the gradually diminishing dulness of percussion, which is much more sharply defined, and terminates more abruptly in cases of enlargement and abscess of the liver. Even Galen was aware of this point of distinction, and by means of it diagnosed the existence of an abscess of the abdominal walls, in a person named Stesianus, in whom other physicians believed there was an abscess of the liver.

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* The Castle Spring (*Schlossbrünn*) is the name given to one of the springs at Karlsbad. See note, page 58.—TRANSL.

b. From diseases of the Parietal Peritoneum.

There are likewise certain cases of circumscribed peritoneal exudations, and of cancer of the peritoneum, which may puzzle our judgment respecting the volume of the liver. The discrimination of the former of these conditions is especially difficult, when the exudation lies in the hollow of the diaphragm and pushes the liver before it. In such a case, when the previous history does not give us any assistance, the diagnosis may be impossible, as I have myself experienced. When, however, as more generally happens, the exudation extends beyond the boundaries of the liver, the nature of the case may be easily recognised from the thinness of the layer of exudation causing a very gradual diminution of the dulness on percussion—a sign which, in several instances, we have found to be a sure guide in distinguishing the exudations which frequently take place in the left hypochondrium, from tumours of the spleen. Exudations of a considerable amount occurring between the liver and the diaphragm produce, also, a bulging of the right hypochondrium, fluctuation in the enlarged intercostal spaces, and paralysis of the diaphragm. Cancer of the peritoneal membrane is in general easily recognised from its irregular distribution.

General or circumscribed accumulations of gas in the peritoneal cavity, such as result from perforation of the stomach, may partially or entirely cover over the liver, and render it inaccessible to percussion. The presence of these accumulations is in general revealed by the equable arching of the abdomen, by the history of the case, and by their being preceded or accompanied by the symptoms of peritonitis from perforation.

c. From Diseases of the small and large Omentum.

These are, for the most part, of rare occurrence, and can, only in exceptional cases, embarrass the examination of the liver. I give two examples from my own experience, in one of which the nature of the affection could not be diagnosed.

OBSERVATION No. II.

Cancer of the lesser Omentum.—Compression of the Portal Vein and Atrophy of the Liver.—Ecchymoses in the serous covering of the intestines and in the parietal peritoneum.

C. Hesse, aged 62, had suffered for some months from increasing emaciation, œdema of the feet, and derangement of digestion. The abdomen was painful, and there was a semiglobular prominence in the right hypochondrium and epigastrium. Here there could be felt a tumour covered with nodules from the size of a walnut to that of an apple, which could be traced as far as six centimètres (2⅜ Eng. inches) below the umbilicus, and which extended into the left hypochondrium. In it there could be felt distinctly fluctuating cysts, surrounded by a dense margin, some of them of the size of half a walnut, or more. The tumour disappeared beneath the ribs, and by means of percussion could be traced upwards as far as the middle of the fourth intercostal space. Its longitudinal diameter in the median line amounted to 32 centimètres (12½ inches). The intestinal canal was full of gas; some fluid effusion could be felt in the lower part of the peritoneal cavity; the spleen was enlarged, reaching to the margin of the eleventh rib.

The tongue was covered with a greyish-yellow coat, there was loss of appetite, and a feeling of fulness after every meal. The bowels were inactive, and the stools of a pale-brown colour. Pulse 60. No jaundice.

The tumour, which could nowhere be isolated from the liver, and which, also, formed part of its contour, was of course regarded as a tumefaction of this organ, and as certainly due either to a cancer containing cysts, or to an hydatid. The latter opinion was counter-indicated by the great tenderness of the tumour upon pressure, the absence of the tremulousness of hydatids, and the firm consistence of most of the nodules, the number of which, moreover, greatly exceeded the usual number of hydatids. A small quantity of the fluid contents was carefully drawn off from one of the cysts, with a fine exploratory trocar. This specimen contained blood and abundance of albumen. It differed also entirely from the fluid of hydatids. Thus, the only tenable hypothesis was that of cancer.

Infusion of rhubarb, with cherry-laurel water and acetic ether, were prescribed. On the following day, November 11th, 1854, in consequence of a copious evacuation, the abdomen was less tense, and the tenderness less; the loss of appetite remained the same. On the 13th, the patient's condition remained unchanged; the exhaustion had increased, and the cysts emptied on the 10th, had become refilled. Pulse small, 72.

On the 16th of November, death supervened under symptoms of œdema of the lungs. Upon *post-mortem* examination, nothing abnormal was found in the cranial and thoracic cavities, except the ordinary changes of old age, and the œdema of the lungs.

In the abdominal cavity there was an abundant effusion of bloody serous fluid; the parietal peritoneum, as well as the serous covering of the small intestine, were covered with numerous ecchymoses, from the size of a lentil to that of a pea, which were so densely studded together, that the serous membrane appeared of a reddish-brown colour. The spleen was enlarged by one-half, and contained much blood. The tumour, situated in the right hypochondrium, extended as high as the lower margin of the third rib. It consisted, for the most part, of a soft, greyish-yellow mass of medullary cancer, with numerous cysts, the size of a walnut, scattered through it, which sprung from the small omentum, covered over the stomach and transverse colon, and penetrated from below upwards (in the form of a wedge) into the parenchyma of the liver. This last was considerably atrophied, and lay deep in the hollow of the diaphragm. The gall-bladder was visible upon the upper surface of the cancerous mass. The portal vessels, especially the right branch, were completely compressed at one part by the cancer. The pancreas, mesenteric glands, kidneys, and urinary-bladder were free from disease.

It is impossible during life to distinguish a new growth of the nature just mentioned, from a tumour of the liver, and the margins of the two pass so insensibly into one another, that we cannot imagine any possibility of isolating the one from the other by means of palpation through the abdominal wall.

Similar, although usually slighter difficulties in diagnosis, may result from cancer of the great omentum. Here, the problem to be solved is in general simplified from the fact, that the nodulated tumours in the great omentum, almost always leave some parts unaffected, through which the clear intestinal sound is transmitted, so that the absence of continuity in the dull percussion sound must prevent our mistaking them for new growths in the liver. But to this there are exceptions, although they are of rare occurrence. In Fig. 16. is represented the position of a cancer of the great omentum, which extending from the liver, filled up the entire abdominal cavity. The thickness of the omentum was so remarkable, that upon percus-

FIG. 16.

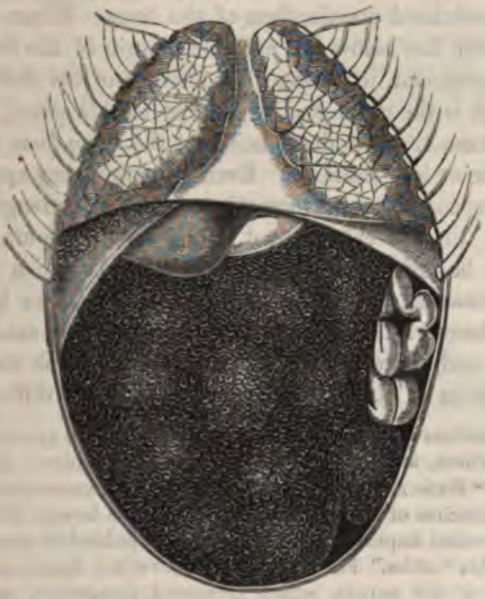

FIG. 16. Cancer of the great omentum, forming a mass which filled up the entire abdominal cavity. The dulness on percussion during life was continuous with that of the liver, and the omentum was so thick that the clear intestinal sound could nowhere be distinguished through it.

sion the intestinal sound could nowhere be detected. Tubercular infiltrations of the omentum also now and then attain a similar thickness. I have found the omentum, from this cause, upwards of an inch in thickness.

d. From abnormal conditions of the Stomach and Intestine.

The great mobility of the intestinal tube, which allows of manifold changes in its situation, not unfrequently embarrasses the examination of the liver in a high degree, and the more so as the determination of the situation of the intestine is rendered uncertain from its contents being sometimes gaseous, and sometimes solid.*

Accumulations of fæces in the transverse colon, if they are limited to the right hypochondrium, may very easily be mistaken for smooth or nodulated tumefactions of the liver. When the portion of colon which lies below the anterior margin of the liver, is filled with solid excrement, it produces an increase of the dull percussion sound, which is the more readily referred to this gland, as not unfrequently from the bile ducts being at the same time compressed, there is more or less jaundice. Even palpation may appear to confirm this supposition, and sometimes even to strengthen it, for in addition to the enlargement, nodulated tumours similar to cancer may be felt. The hard knotty scybala which exist under such circumstances may easily be mistaken for cancer by the inexperienced observer. Cases of this nature are met with most frequently in women; and, were it necessary, I could furnish the details of an entire series of them.† In general, it is not difficult to avoid mistakes in diagnosis arising from these causes. Swellings due to accumulation of fæces are seldom confined to the ordinary boundaries of an enlarged liver; they pass beyond this on one side or another, in such a manner as could not be imagined to take place from increase in the volume of this gland. The abdominal parietes are not too thick to prevent our distinguishing the peculiar doughy feel, upon palpation, furnished by fæcal matter, from the firmer consistence of the liver. In doubtful cases, we must not forget the valuable rule, to

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* The situations of the large intestine, in relation to surrounding parts, are very various, and are hence difficult to determine. Long ago, De Haen, in his "*Ratio Medendi,*" took notice of this circumstance, and gave a number of sketches of abnormal situations of this bowel, which appeared to him of practical importance. Annesley also published some interesting examples in his "*Atlas.*" For some years I have had drawings made of the appearances of this nature, which presented themselves on *post-mortem* examination, and I shall bring forward the most important of these in a subsequent memoir upon diseases of the large intestine. This circumstance claims our consideration, not only in diagnosis, but also in reference to many other points.

† One example is here annexed, which is interesting from the fact that a double displacement had taken place. A female, aged 25, living in the country, who had already aborted several times, believed that she was in the family-way, owing to the cessation of menstruation, the presence of

squeamishness, &c. The ordinary medical attendant enjoined the strictest rest, which, from her anxiety to avoid, at any price, a fresh abortion, she maintained by lying for six months upon a sofa. A vaginal examination was not permitted; it was only by feeling the abdomen, that the medical man recognised a rounded tumour rising up out of the pelvic cavity, and reaching by degrees to the umbilicus. Meanwhile, the anxiously-expected movements of the child did not make their appearance; and, notwithstanding the most careful nursing, the young woman fell away, became of a pale-yellow colour, lost her appetite, got œdema of the feet, and at length complete jaundice. A second medical man who was called in, declared that the disease was an enormous swelling of the liver, and denied the existence of pregnancy; in opposition to which, the first medical man urged the fact (which he had observed) of the tumour growing upwards from below. On my opinion being asked, I examined the abdomen more closely. It was remarkably distended and tender; a tumour was seen rising up from the left side of the cavity of the pelvis, which felt doughy, and at the umbilicus extended 1½ inch beyond the median line; the cæcal region yielded a clear tympanitic sound as far as the linea alba. The hepatic dulness, in a line with the mamma, extended from the fifth rib to 8 centimètres (3¼ inches) below the arch of the ribs, but in the axillary line did not pass beyond the margin of this arch. Transversely, through the epigastric region, there ran a cylindrical swelling, which was tender upon pressure, and which yielded, upon percussion, at some places a clear sound, and at others a dull one. The bowels were moved every second day, and the colour of the stools varied, being sometimes pale and sometimes dark. Hence, I expressed my opinion that pregnancy did not exist (an opinion which was contradicted by the form, and more especially by the doughy consistence of the swelling, which could have only arisen from an unusually long sigmoid flexure distended with fæcal matter), and that the condition of the liver could only be judged of after the evacuation of the intestinal canal. By means of clysters, and compound infusion of senna, an extraordinary amount of fæces was evacuated. After eight days, it was reported to me that the lower tumour had disappeared, that the liver was much smaller, and that the jaundice had diminished. Three weeks later, when the patient presented herself to me, after having drunk of the Kreutzbrunnen Springs of Marienbad, no enlargement of the liver was any longer to be detected;—by means of purgatives she had lost her hope of a child, and, at the same time, her anxiety about a diseased liver.

clear out the intestinal canal before examining the liver, and not to be deterred from this, by the statements of the patient, to the effect that he has a daily evacuation, or occasional diarrhœa, because considerable accumulations may be present notwithstanding.

The intestinal canal sometimes embarrasses the examination of the liver in another way, namely, from folds of the large intestine, or more rarely of the small, being interposed between the gland and the abdominal wall. In most cases, it is the first curve of the colon which is pressed forward, and lies upon the upper surface, sometimes of the right (Fig. 17.), and sometimes of the left lobe.

FIG. 17.

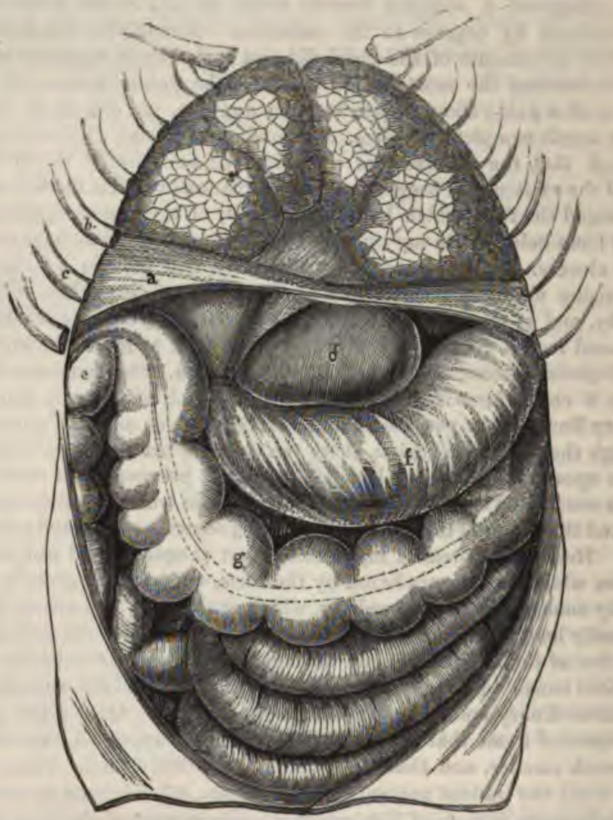

FIG. 17. Represents a case in which the right lobe of the liver was found overlapped by the first curve of the colon.

One must suspect such a superposition of the colon upon the liver, when the diameter of the liver at one part is found, upon percussion, to be unusually small in comparison with that at an adjoining part. In many cases, almost the entire length of the colon lies over the liver, so that a determination of the size of this organ is impossible. (Fig. 18.)

FIG. 18.

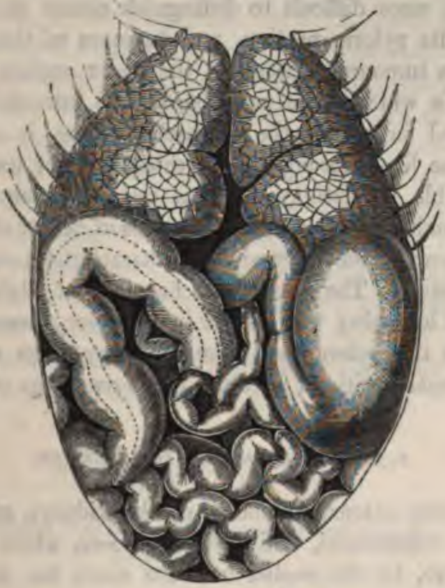

FIG. 18. Represents a case in which the entire liver was covered by the colon.

The large intestine is sometimes of such unusual length, as to assume situations which could scarcely have been expected. I have before me the sketch of a distribution of the parts which I met with some days ago in the *post-mortem* examination of a man, 33 years of age; the middle part of the liver is represented as covered by the fold of the sigmoid flexure, whilst the lateral parts are hidden by the transverse colon lying above this fold. Such displacements of the intestine are usually only temporary. There are cases, however, in which they continue for months, and finally lead to atrophy, and to the formation of depressions upon the upper surface of the gland.*

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* An observation of this nature was made in this Clinique, in the case

The stomach contributes more rarely than the intestinal canal to the embarrassment of the examination of the liver. The obstacles which a stomach distended with food opposes to the determination of the size of the left lobe of the liver, are easily avoided, if a proper time be selected for the examination. When the stomach is completely empty, the left lobe, as has already been observed, falls somewhat downwards.

It is much more difficult to distinguish cancer of the stomach, especially of its pyloric portion, and tumours of the head of the pancreas, from tumours situated on the under surface of the liver. Cases occur in which these parts become so intimately united by adhesions, and in which the diseased pylorus is so completely covered by the liver, through the greatest part of its extent, that it is impossible to distinguish them by either palpation or percussion. In such cases, the other symptoms of cancer of the pylorus, the enlargement of the stomach, the characteristic vomiting, &c., decide the matter. The jaundice is here but of slight importance as a symptom localising the disease in the liver, because cancerous exudations in the pylorus and pancreas very often close up the *ductus choledochus*, without affecting the parenchyma of the liver.

e. From Diseases of the Kidneys.

It is only very extensive tumours of the kidneys, such as medullary cancers, echinococci, and hydronephroses, which come to be considered here, for the smaller ones are much too deeply situated to be confounded with hepatic affections. In most cases, the diagnosis is facilitated by the fact that enlargements of the kidneys are covered by folds of intestine containing gas, which is not usually the case with those of the liver. According to my experience, the swelling which depends upon large cancers of the kidneys, is, in most cases, situated in the cæcal region, in contact with the abdominal wall, and is then separated from the liver by portions of bowel. I only remember one patient in whom this was not the case, and in whom cancer of the kidney had been mistaken for cancer of the liver. The tumour of the kidney, as large as a child's

of a man, 48 years of age, in whom, during three months, a fold of the colon covered over the left, and part of the right, lobe of the liver. The displacement, which was made out during life, was confirmed by *post-mortem* examination.

head, was in contact with the under surface of the liver, and had pushed this organ upwards almost an entire intercostal space. It was not covered by coils of intestine containing gas. On the patient's back it was easy to pass the finger into a space between the swelling and the margins of the rib, a sign which, as Bright has justly observed, contra-indicates cancer of the liver. In the case just mentioned, the tumour could only be mistaken for cancer of the liver, by assuming that there was a new growth projecting, in an isolated manner, from the posterior part of the liver. But such growths never protrude so far from the parenchyma of the liver, in which they lie embedded, as must have been assumed to be the case in this instance. When tumours of the kidneys are present, we must not calculate upon finding, upon that account, a prominence in the lumbar region close to the margin of the quadratus muscle, which may serve for distinguishing them from hepatic affections; such a prominence is usually sought for in vain, because tumours of the kidneys, in their development, take chiefly a direction forwards and downwards.

The following instructive case may farther illustrate the relative situations of these organs in disease.

OBSERVATION No. III.

Cancer of the Right Kidney.—Displacement of the Liver upwards and to the left.

J. Rother, a boy aged 16, a pupil in the Blind Institution, after having been treated for a long period in the surgical division of the Hospital, was removed, on the 19th of August, 1856, to the medical department. For three weeks before, there had been observed a tumour, rapidly increasing in extent, which projected from under the right arch of the ribs, and was covered with enlarged cutaneous veins. It was smooth and elastic, appeared to fluctuate in some places, and was notably displaced upon a deep inspiration. Percussion in the mammary, as well as in the axillary lines, yielded a dull sound, extending 21 centimètres (8¼ inches) downwards from the third rib. This dull space stretched backwards as far as the sixth dorsal vertebra; it could be traced beyond the epigastrium as far as the axillary line on the left side, and upwards as high as the fifth left rib; the lower intercostal spaces of the left side were widened.

The impulse of the heart was felt between the third and fourth ribs. Pulse 120 to 130; respiration free; appetite moderate; stools normal; urine pale, and containing neither albumen nor blood.

The boy became rapidly anæmic, so that death ensued as early as the 24th of September. Upon *post-mortem* examination, the liver appeared remarkably enlarged; it filled up both the hypochondria and the epigastrium, as far as the umbilicus; the diaphragm was pushed upwards by it as high as the third rib on the right side, and on the left side as high as the lower border of the fourth ribs. At the same time, the organ was compressed on the right side, so that the suspensory ligament lay in the left mammary line, and the gall-bladder to the left of the linea alba. Upon closer inspection, it was found that the apparent enlargement of the liver depended upon an enormous cancer, which sprung from the right kidney, and had compressed the right lobe of the liver so as to reduce this to the thickness of a mere membrane. This tumour was bounded externally by a firm capsule, and consisted of a white mass of medullary cancer interspersed with numerous apoplectic patches. The tumour weighed 8.7 kilogrammes (19 pounds avoirdupois). Close to the suspensory ligament, there were embedded in the substance of the liver a couple of cancerous nodules, the size of beans. Similar secondary deposits were found in the upper lobe of the left lung; the lower lobe was compressed. The heart was moved upwards, but otherwise normal; the spleen was anæmic, and of moderate size; the stomach and intestinal canal exhibited no important structural change; the left kidney was hypertrophied.

It is not my present object to enumerate all the causes of mistakes in diagnosis, which may throw uncertainty over the results of clinical examinations of the liver. I wish merely to communicate those which my experience in practice, and in the *post-mortem* theatre, has pointed out to me as the most important and the most necessary to be borne in mind.

He who wishes to master this department of the diagnostic art, so far as it is practicable,* or even he who would avoid with certainty the commission of great blunders in diagnosis, must study the relative situation of the abdominal viscera in their pathological conditions, have great experience in *post-mortem* examinations, and neglect no opportunity of rectifying and extending his clinical knowledge upon this head. The practice of determining with accuracy and certainty the boundaries of solid tumours in the abdominal cavity, by means of the pleximeter, is the school for this work; but he who thinks that this will suffice, must not be surprised if his diagnosis be frequently corrected by *post-mortem* examination.

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* I have not considered here, displacements of the liver by aneurisms of the aorta, or of the hepatic artery, or by retroperitoneal tumours, because I have met with no complete case of this nature, that is to say, in which there was an opportunity of following it up by *post-mortem* examination. What has been already communicated must suffice for correcting our diagnosis under such circumstances.

CHAPTER IV.

JAUNDICE.

(*Icterus, Aurigo, Morbus Regius, Morbus Arquatus.*)

1. *Historical Account.*—The yellow-tinging of the skin, and of several of the secretions by bile-pigment, has always greatly engaged the attention of physicians. Accompanying this condition we meet with a series of diseased processes, which, under the names of icterus, bilious fever, bilious conditions, &c., even in the infancy of medicine, and from that period through all ages down to our own day, have been studied sometimes more zealously, and at other times less so, according to the prevailing constitution of disease, and local influences. Copious materials have accumulated in the course of time,* and theoretical views of various natures have started up and fallen to the ground, without the most important fundamental questions having received a satisfactory explanation.

Passing over the detailed history of the subject, we meet with two principal theories concerning the different forms of disease under discussion, which we shall, in the first place, treat of collectively, because the distinctions between certain varieties of jaundice and bilious fevers † cannot everywhere be accurately defined. Of these two theories, sometimes the one has found its supporters, sometimes the other, and not unfrequently both at the same time.

1. One of these theories is, that the seat of these diseases is to be looked for in a disturbance of the functions of the liver; that they arise from abnormalities in the secretion, or in the excretion of bile; and that thus they exhibit symptoms indicative of derangement of the liver.

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* See Eisenmann, Krankheitsfamilie Cholosis. Erlangen, 1836.

† In order to circumscribe somewhat the extremely vague collection of morbid processes formerly designated by this name, we include here only those forms which are accompanied by a jaundiced-tint of the skin and of the excretions.

During the time of Hippocrates, and down to the decline of the doctrines of Galen, bile secreted in excessive quantity, or of bad quality, was regarded as a fruitful source of diseases; its overflow into the body, its passage from the abdomen into the blood, and its abnormal colour, were looked upon as so many etiological impulses, from which numerous disturbances took their origin. The cause of these anomalous conditions remained undetermined; persons were contented with collecting observations, and bringing them into harmony with the predominant opinions of the day.

When, in the sixteenth century, the views concerning the functional importance of the liver began to assume another form, these theories underwent a change, and, as usually happens, passed over almost to the opposite extreme. Paracelsus and Van Holmont rejected almost all the pathogenetic properties of the bile; the former, on the ground that this secretion was nothing more than a useless refuse; the latter, because he thought it impossible that such an excellent fluid, the very balsam of life, could give rise to disease.

This change of opinion, however, had but a short duration, and was never generally recognised. Even Sylvius * again, regarded the bile as an important source of disease, in a similar manner to the ancients, except that the theory of its mode of action was transferred to a chemical basis. The opinions concerning jaundice and the allied affections were so far cleared up, that a disturbance in the evacuation of the secretion formed in the liver was always distinctly recognised as the cause of an excess of bile in the blood, the more the zeal for anatomical studies increased. The question as to whether there could be a suppression of the secretion was sometimes affirmed and sometimes denied, according as the liver or the blood was regarded as the seat of the secretion of bile. The supporters of the latter view, who, like Glisson, looked upon the liver as only a peculiar organ for filtering the bile from the blood (*peculiare collatorium*), who maintained that interruption of the functions of the organ was the cause of bilious conditions of the system, and who included among them such weighty names as those of Morgagni, Boerhaave, Van Swieten,† and others, gradually lost ground in opposition to those who, like

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* Opera Omnia, p. 469.

† Comment. in H. Boerhaave, Aphor. III. 127. Semper autem supponit (icterus) vel impeditam secretionem à sanguine venæ portarum vel impedimentum tollens liberum exitum bilis secretæ in duodenum.

Monro,* Eller,† Werlhof, Selle,‡ Reil,§ &c., believed that jaundice arose only from an obstruction to the excretion of bile. The arguments, which at that time were urged against jaundice being the result of a suppression of the functions of the liver, have been collected in Marcard's "*Medicinischen Versuchen*" (*Part I.* p. 12).

The same results were arrived at by W. Saunders,‖ who was the first to investigate experimentally the passage of the bile into the blood.

In more recent times this theory has acquired a still firmer foundation, since the chemical and physical properties of the bile have begun to be examined more carefully, and since the constituent elements of this secretion have been sought for in vain in the blood of the portal vein, and likewise of other vessels. It became, therefore, necessary in all cases and forms of jaundice to search for causes, which could obstruct the evacuation of the biliary ducts. For most of the forms of jaundice, this could be done without difficulty; but in other cases, no material proof of such obstructions could be furnished, however readily catarrhal constriction of the ducts was assumed to exist; even the spasmodic contractions and paralysis to which they were supposed to be liable, could not always suffice.

Such forms consequently remained unexplained, seeing that the experiments of physiologists were not taken into account, and as there was an unwillingness to adopt any new theory. The consequence has been, that a large proportion of physicians, such as Darwin, Andral,¶ H. Mayo, Watson,** Budd,†† &c., have maintained that the bile does not originate in the liver, but is previously generated in the blood, and that thus, anything which interferes with its elimination may give rise to jaundice. Even in Germany opinions have recently been expressed in favour of this view, although the repeated chemical analyses of the blood of the portal vein, as well as the experiments of extirpating the liver, have thrown a new light upon the secreting function of this organ. Many pathologists, however, reject both theories.

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* Account of the Diseases of British Military Hospitals, p. 206.

† De cognosc. et curand. morb., p. 221. Icterum oriri non posse nisi bilis prius a sanguine segregata ejusque circulo deinceps commista.

‡ De curand. hom. morb., Ed. Sprengel, p. 184. Secretionis bilis impedimenta icterum efficere nequeunt, quum bilis secretione producatur.

§ Icterus est bilis jam in hepate secretæ redundantia in sanguinem ex qualicunque excretionis impedimento.

‖ Abhandlung über die Structur der Leber. Leipzig, 1795, S. 60.

¶ Bullet. des Sciences méd. 1816.

** Prakt. Heilkunde übers, von Steinau. Bd. IV., S. 264, ff.

†† Diseases of the Liver, p. 374. "In a large proportion, perhaps in the greater number of cases, jaundice results primarily and solely from the secretion of bile being suppressed or deficient."

2. The second theory is, that under morbid conditions of the system, substances are formed in the blood without the co-operation of the liver, which in colour and other properties resemble the ingredients of bile, if they are not identical with them; but which only assume a pathological importance from their quantity being in excess.

This view as to the formation of bile, if we except obscure hints at it, already existing in the writings of Galen, Sydenham, Baillou, &c., was first clearly announced by Bianchi:* "Sunt duo primaria icteri genera, primæ classis icterus a vitio hepatis, alterius speciei icteritia a causa solutiva sanguinis." The same view was fully discussed by Grant,† who assumed that the yellow material of the serum consisted of bile, and who attributed the causes of bilious diseases to an excessive quantity, or to alterations in the quality of this fluid, quite independent of the liver.

This theory was also maintained by Reil in his "*Tractatus de Polycholia,*" published in 1782; ten years later, however, he retracted his opinion, in the "*Memorabil. Clinic.*" (*Fasc. IV.* p. 48), in which he attributed the change of the fluids in question to an abnormally increased activity of the liver: "Nunquam nisi hepatis ope, neque bilis, neque ipsius analogon conficitur."

The ideas of Grant were reiterated in the observations published by J. P. Schotte,‡ "*On the Malignant Fever on the Coast of Senegal.*" A. Diel § likewise attributed the cause of bilious diseases not to the bile *already formed,* but to an increase of the elementary constituents of this substance in the unhealthy juices of the body.

Senac,‖ also, thought it probable that the red portion of the blood was the peculiar material of the bile, and that it assumed a yellow colour when it became putrid or otherwise decomposed.

More recently, since the subject of pigments has been more carefully studied, and since the doctrine that the hæmatine of the blood forms the basis of all pigments, has become more generally accepted, there has been no lack of observers who, adopting the ideas of Senac, have referred the jaundiced-tint of the skin, which is present in pyæmia, in putrid infection, and other allied conditions, without any morbid condition of the liver, to a direct metamorphosis of the hæmatine into a yellow substance similar to, if not identical with, bile-pigment. Breschet, to whom belonged the merit of having first supported the derivation of the pigment from the blood by direct proofs,* bluntly expressed this view as to the origin of jaundice as follows :—

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* *loc. cit.*, pp. 75-78, 185, 313.

† Observations on Fevers, vol. I., p. 30.

‡ Von einem austeckenden schwarzgallichten Fieber, welches im Jahre 1778 am Senegal herrschte. Aus dem Englischen. Stendal, 1786.

§ Baldinger's neues Magazin. Bd. 7, Stück, 5.

‖ De recondita febrium natura, p. 25.

"Je presume ainsi, que l'ictère est occasionné bien moins par la "bile que par le sang."

A similar expression is made use of by Dubreuil †:—"La teinte "ictérique est la suite d'une modification maladive des parties con-"stituantes du sang, peut-être de la matière colorante portée sur le "serum."

This theory has acquired fresh support from the investigations of Virchow upon pathological pigments. ‡ These investigations have proved that, under certain circumstances, a yellow substance is formed from the hæmatine which, in its relation to solvents and reagents, bears a close resemblance to cholepyrrhin.

Zenker and Funke § have brought forward fresh arguments in favour of the intimate relation subsisting between the bile-pigment and the red matter of the blood, by showing that a modification of the colouring-matter of the bile, viz., bilifulvine, can easily be transformed into "hæmatoidine,"—a derivative of "hæmatine." This would appear to indicate the possibility of a direct transformation of hæmatine into cholepyrrhin.

We shall subsequently see that the observations upon the formation of pigment, however numerous these may be, are not yet sufficiently mature for arriving at any conclusion; but that there exist still other sources of colouring-matter hitherto neglected, which may assign to the brown matter of the bile another place in the series of pigment changes.

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* Considérations sur une altération organique appelée dégéneration noire. Paris, 1821. Journ. de Magendie, Tom. I.

† Ephémér. medic. 1826.

‡ Virchow, Archiv. fur patholog. Anatomie. Bd. I.

§ Lehmann, Lehrb. der physiol. Chemie. Bd. I., S. 292.

The two theories of bilious affections upon which some historical light has just been thrown, have at no time received a universal application. They were employed to explain the origin of those forms of jaundice only, in which no proof existed of any obstruction to the escape of bile. There has been no want of opposition to these theories. I shall content myself by mentioning here the nature and manner in which J. P. Frank, in his time, expressed himself in regard to them :—*

"Ita quoque cutis flavedinem in ictero non tam a bile resorpta "quam a cruoris ipsius in vasis metamorphosi aut combinatione, si Diis "placet, chemica aliqui derivant. Sic, eheu! hypothesis in rebus medi- "cis hypothesin trudit; et montem, qui præoccupatos ante oculos "prominet, in acervis formicini favorem, arenæ pro grumulo declarat."

In addition to the theories just discussed, others have been brought forward from time to time, which require no detailed critical examination. To these belong the view frequently entertained by the ancient physicians, that the bile was infiltrated into the tissues, owing to its becoming fluid from the operation of poisons, marsh miasma, putrid substances, &c.;—Regner de Graaf (*De succo pancreatico. Cap. VIII.*), Godart (*Journal de Médecine. Tom. XVIII.*, 1763).† Among these, also, may be included the view, which made jaundice depend upon a spasmodic condition of the skin, and an obstruction to the circulation of blood through it; and, lastly, the theory of Deyeux (*Thèse de Paris*, 1804), and of Gaultier, (*Thèse de Paris*, 1811), who imagined that the yellow colouring-matter was secreted by the skin, without any necessary participation of the liver.

2. *Theory of Jaundice.*

There can be no doubt that most, although not all, cases of jaundice, are produced by the re-absorption of bile already secreted. In general, it is not a difficult matter to point out the anatomical nature of the mechanical obstruction to the excretion of bile; and numerous instances in which the experiment of applying a ligature to the ductus choledochus was performed, years ago, by Saunders, Tiedemann, Gmelin, and many others, have thrown some light upon jaundice resulting from the passage of the secretion into the lymphatics and veins.

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* De curand. homin. morbis epitome. Vienna, 1821. Lib. VI., Pars 3, p. 336.

† Pliny had long before imputed a case of jaundice to a great liquefaction of the bile.

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Jaundice from re-absorption, therefore, forms the sure starting-point for further pathological inquiry, and in all cases and forms of the affection where it is practicable, the main question is to search for mechanical obstructions preventing the escape of the bile, or for other causes of the passage of this fluid into the blood. It is only when this is impossible, that we can consider other theories of which a positive confirmation has hitherto been impossible, and the main value of which consists in the necessity for some hypothesis for explaining our observations. In such cases can we ascribe the jaundice to an accumulation of bile in the blood, owing to something which interferes with its secretion, or are we to adopt the theory of a direct crumbling down of the blood corpuscles, or red matter of the blood, into bile-pigment?

The production of jaundice from an imperfection in the secreting functions of the liver, which Budd and Bamberger* have spoken of in recent times, without, however, bringing forward any striking proofs of the assertion, is opposed by too many well-established facts for us to support it. All the means for detecting traces of the essential elements of the bile in the blood generally, and in that of the portal vein in particular, have been exhausted without any result; neither the colouring-matter, nor the acids of the bile,† substances for which we possess tests of considerable delicacy, have been found. Even if it be granted that the quantity of bile which circulates with the blood under normal circumstances is immeasurably small, owing to the constant performance of the functions of the liver, in the same way as occurs in the case of the urine,‡ still this argument is of no weight in diseased conditions, in which the disorganisation of the glandular parenchyma limits the secreting function, or arrests it to a greater or less degree. In the same way that urea accumulates in large quantity in the blood in granular degeneration of the kidneys, so ought the biliary acids and bile-pigment to accumulate in the blood in cases of granular liver. Repeated observations have proved that this is not the case. Further on I shall relate a case of disease (No. XXIX.), where the secretion of bile was completely arrested in consequence of fatty degeneration of the liver, so that the contents of the bowels were found pale, the gall-bladder empty, and the biliary ducts coated with a greyish mucus, notwithstanding which, the skin was of a chalky paleness, and the urine not tinged with bile-pigment.* Still more decided are the proofs of the formation of bile in the liver, which have been furnished by the beautiful investigations of Müller and Kunde,† and likewise of Moleschott.‡ In the case of frogs which were kept alive for two or three days, and by Moleschott for several weeks, after being deprived of their livers, not a trace of the elements of the bile could be detected in the blood, the lymph, the urine, or the musuclar tissue.

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* Handb. der Pathologie und Therapie von Virchow. Bd. VI., p. 518.

† From the researches of Strecker, the bile would appear to contain two resinous acids, the Cholic (*Strecker*), or Glycocholic (*Lehmann*), and the Choleic (*Strecker*), or Tauro-cholic (*Lehmann*). According to Lehmann, these acids are formed by the "conjugation" of cholic acid with Glycine (gelatine-sugar) and Taurine respectively; and they are united in the bile with soda as a base. Lehmann makes the composition of Glycocholic acid $C_{52}H_{42}NO_{11}HO$, and that of Taurocholic acid $C_{52}H_{45}NS_2O_{14}$.—Transl.

‡ If we assume, what may not be far from the truth, that 1 kilogramme (2.2046 lbs. avoird.) of bile, containing 6 per cent. of solid matter, is secreted daily by an adult man, the quantity of the acid of the bile contained in the blood must be twice as great as that of the urea, the daily quantity of which is calculated at 30 grammes (463 grains Troy). But Pettenkofer's test indicates the presence of a smaller quantity of the acids of the bile than any test does for urea. Accordingly, the detection of bile in the

The idea of the pre-existence of bile in the blood is further opposed by the arguments in favour of a conversion of certain elements of the blood into bile within the liver, which may be derived from the different composition of the blood in the portal and hepatic veins. Although the difference of the blood from these two sources indicated by Lehmann's analyses, appears almost too great to be explained by the act of secretion, it, nevertheless, opens up interesting points for studying the origin of bile. We shall, hereafter, have an opportunity of adducing fresh data for obtaining a deeper insight into the mode of origin of the hepatic secretion, by showing how certain products of metamorphosis disappear within the liver.

Bamberger considers that he has found a proof of the pre-exist-

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blood ought to be an easier matter than that of urea, a circumstance which must increase the value of the above observation.

* Haspel (Maladies de l'Algérie, Tom. I., p. 262) observes:—"J'ai eu "l'occasion de constater plusieurs fois l'absence d'ictère dans les cas de "destruction presque complète de l'organe hépatique. La bile n'était "plus secretée, la vesicule ne contenait qu'un mucus blanchâtre."

† Kunde, Dissert. inaug. Berol. 1850.

‡ Archiv für physiol. Heilkunde. Bd. II., S. 479, ff.

ence of bile in the portal vein in the jaundice, which is observed to accompany inflammation of that vessel. The blood of the vena porta, which passes by the collateral vessels into the general circulation, it is thought, in such a case, produces jaundice by the spontaneous elimination of the elements of bile. During the past year, I have met with three cases of complete obstruction of the portal vein, and have availed myself of the opportunity to examine carefully the clot (*Thrombus*) taken from the vessel. Leucine could be detected in it, but not a trace of bile-pigment. Moreover, jaundice is pretty frequently absent in cases of obliteration of the portal vein.* The conclusions which the same author draws from the presence of biliary matter in the fluid of ascites accompanying cirrhosis of the liver, will subsequently receive a simpler interpretation; as will also the gall-stones, which have been observed in the interior of the portal vein by Realdus, Columbus, and Deway.

As regards the second hypothesis, the spontaneous conversion of the red matter of the blood into bile-pigment in putrid diseases, no objection can be made to the possibility of such an event, considering the intimate relations which have been shown to exist between hæmatine and cholepyrrhin; but positive proofs of it are entirely wanting. As yet no one has succeeded in manufacturing bile-pigment from the red colouring-matter of the blood, although the products of decomposition of both are the same. The yellow matter, however, which in cases of pyæmia circulates with the blood, and is voided in the urine, is, at least in most cases, identical with bile-pigment, and participates in all the properties of this substance. But even were the conversion of hæmatine into cholepyrrhin, under the operation of certain agencies, possible, still it remains to be proved that this metamorphosis really takes place in the course of putrid diseases within the blood-vessels of the living body. As to the absence of pigment in the interior of the hepatic cells as an argument against the theory of re-absorption in consequence of obstruction, which has been urged, particularly by Virchow, † it is of no weight, provided ways can be shown in which accumulations of pigment may take place in the blood without any obstruction to the escape of the bile.

If, after what has been said, we should have legitimate scruples in making use of hypotheses, which rest upon an uncertain foundation, the question comes to be, how those cases of jaundice are to be explained which occur without any remarkable obstruction to the excretion of bile. The number of such cases is not small, for they include, in addition to the jaundice of pyæmia and of the conditions allied to this, those forms of jaundice which result from the narcotism of chloroform and from other forms of intoxication, and lastly, those which are met with in many instances of pneumonia, in intermittent fever, in the bilious fevers of marshy districts, in inflammation of the portal vein, &c.; we likewise refer to this class the jaundice induced by mental affections, for which spasm of the bile ducts—the cause to which it is usually referred—offers no satisfactory explanation.

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* See Gintrac. Sur l'obliteration de la veine-porte. Bordeaux, 1856.

† Virchow, Archiv f. path. Anatomie. Bd. I.

In order to arrive at some clear ideas upon this point, so far as the present state of circumstances permits, we must examine more closely the causes which can give rise to an accumulation of bile in the blood. These may be said to be twofold, provided the formation of bile is in the first place assumed to be constantly the same.*

I. Through increased absorption of bile from the liver into the blood.

II. Through small consumption, or a sparing change of the bile, which has been taken up into the blood.

We leave the latter unconsidered at present, and occupy ourselves in the first place with—I. *The conditions upon which the passage of a large quantity of bile into the blood may depend.*

The mechanism of biliary obstruction is much more complicated than it is considered to be by pathologists who usually only pay attention to the condition of the biliary ducts. Obstructions may exist during life, which are with difficulty accessible to the knife of the anatomist, and which, to a certain extent, cannot be demonstrated. It is known that two products of cell-growth, bile and sugar, are simultaneously produced in the liver; the latter passes into the blood of the hepatic vein, the former into the commencement of the bile ducts. The current towards the blood can only result from diffusion, that occurring at the same time towards the bile ducts, from filtration. It is difficult to determine how the separation of the two currents is possible; why the contents of the cells surrounded by capillaries almost like a ring should be distributed partly in one direction, and partly in the other. We must, as Ludwig † has with justice acutely observed, either assume that the rapidity of diffusion into the blood of the elements of bile is less than that of sugar, or that the sugar has attractions for some one element in the blood which is wanting to bile. The latter view is highly improbable, for we are acquainted with no substance in the blood for which sugar possesses a pre-eminent affinity. In the former case, the separation of the two substances would not be complete, some bile would always pass into the blood along with the sugar, and some sugar into the bile.* But, whatever view we adopt as to the mode of separation of the substances secreted by the liver, still it is certain, that an increased absorption of bile into the blood may be dependent upon a difference in tension of the contents of the hepatic cells and blood-vessels. Such a condition may arise in two ways: 1, from obstruction of the bile ducts by which the pressure on the side of the cell contents is increased, and 2, from obstruction to the flow of blood in the portal system, and consequent diminution of the pressure on the side of this fluid.

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* We shall subsequently become acquainted with Polycholia, or an excessive secretion of bile, as a cause of several forms of jaundice.

† Physiolog. des Menschen. Bd. II., S. 232.

The former mode of production is the more common, and is the only one which as yet has been investigated. All mechanical obstructions which interfere with, or arrest the flow of bile into the larger or smaller biliary ducts, produce jaundice in this way. A large proportion of these obstructions are of an anatomical nature, and may be detected on dissection, and hence are accurately known; others, however, the existence of which has been maintained, are less clear in their nature, and require further examination.

The flow of bile through the hepatic ducts is principally owing to the *vis à tergo* of the secretion constantly pressing onwards; the anatomical element, muscular tissue, necessary for the assumption of the existence of contractility as the exciting cause, does not exist. Muscular tissue can only be detected in the gall-bladder, and in the cystic and hepatic ducts, and can, therefore, only be taken into consideration in reference to these parts. Muscular contraction was resorted to ages ago for the explanation of many forms of jaundice, and one form of the affection was named *icterus spasmodicus*, which was thought to arise from exposure to cold, mental emotions, &c. To this view it is justly objected, that a closing up of the large bile ducts (and it is only in these that muscular fibres capable of giving rise to spasm exist),† does not produce jaundice until after three days, and that a spasm lasting for three days, without the operation of any persistent local cause, such as a concretion, is beyond the reach of imagination; moreover, the jaundiced tint appears much earlier than is compatible with such a theory. At first sight, it seems more probable, that there might be paralysis of the bile ducts, as was long ago assumed by Galen and Darwin, and for which arguments have been adduced more recently by Frey,* Henle,† and von Dusch.‡ It is doubtful, however, whether paralysis of the hepatic and cystic ducts, can really produce such a degree of obstruction as to result in jaundice, while the other agencies concerned in the propulsion of the bile continue in operation. Moreover, no one has brought forward proofs of the existence of so limited a muscular paralysis without any anatomical lesion: and from what we know of muscular action in the other abdominal organs, such a phenomenon would be quite exceptional. In order to obtain more precise information, I have, in conjunction with my colleague, Reichert, divided both the splanchnic nerves of a cat, and extirpated the largest part of the cœliac ganglion according to the method proposed by Ludwig. The animal lived three and a-half days after. Upon *post-mortem* examination we found the liver congested, and the gall-bladder moderately distended. The mucous membrane of the stomach was congested, and presented, at one part, an ulcer of a rounded form, the size of a groschen (somewhat smaller than a sixpence), and having its margins suffused with blood. There was not a trace of jaundice. Division of the spinal cord both above and below the cervical plexus, in the manner practised by Bernard, has been equally unsuccessful in producing an obstruction to the flow of bile. These experiments were made repeatedly by Dr. Valentin, yet always with negative results.

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* According to Stockvis (*Bijdragen tot de Kennis der Suikervorming in de lever*, p. 35) sugar is always present in the bile; I have myself detected it in considerable quantity, in many cases.

† The older physicians transferred the seat of spasm partly to the mus-

The compression which the biliary organs undergo from the narrowing of the abdominal cavity consequent upon the respiratory movements, has a much greater influence over the flow of bile. This circumstance was already known to the older physicians. Boerhaave remarks: "Bilis vix movetur, nisi aliunde urgeatur, neque protruditur nisi respirationis efficacia." § Bidder and Schmidt || also state, that

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cular tissue of the duodenum, the contraction of which was thought to close the mouth of the ductus choledochus.

* Archiv für physiol. Heilk. Bd. IV., 49. † Rationelle Pathol. II., 195.

‡ Untersuchungen und Experimente als Beitrag zur Pathogenese des Icterus. Leipzig, 1854.

§ Prælect. acad. in propr. institution. Ed. Haller, Vol. III., S. 186.

|| Verdauungssäfte und Stoffwechsel. S. 210.

in all their experiments, they had repeatedly made the observation, that the respiratory movements exercise a powerful influence over the propulsion of the bile which has been secreted. Although it might be difficult to show, that the want of this influence is sufficient to give rise to jaundice, still this must be borne in mind as a co-operating cause. Jaundice is not unfrequently observed accompanying right diaphragmatic pleurisy and perihepatitis of the convex surface of the liver, in which there is certainly a limitation of the free action of the diaphragm, but in which no deeper lesion of the liver or of the bile ducts is to be detected.

Hence it follows, that in general we must be very cautious in assuming causes of obstruction of bile, for which, as in the case of paralysis of the bile ducts, no anatomical origin can be detected.

As regards the second mode in which the bile is made to enter the blood, viz., the obstruction of the circulation of the blood, and the diminution of the pressure exerted on the side of the capillaries of the portal vein, it is estimated with difficulty on account of our comparatively small knowledge of the complicated circulation of blood in the interior of the liver; still it is easy to show that the tension of the stream of blood must be here liable to many variations. There can be no doubt, that from obstruction of the main trunk or of the larger branches of the portal vein, the tension of the capillary vascular system in the liver is diminished, and the entrance of the biliary contents of the hepatic cells into the blood is facilitated. Hence jaundice is of frequent occurrence under such circumstances.‡ Similar conditions result from obstructions of a large number of the interlobular veins of the liver by means of pigment granules, which is a matter of frequent observation in malignant intermittents. An obstruction to the flow of blood in the liver is here produced; at one part the pressure of the capillary vessels is diminished; whilst at another, their tension is greatly increased. In such a case we very often find albumen in the bile as well as jaundice, which last only attains a high degree when the majority of the capillary vessels are blocked up. A diminution of the tension of the hepatic vessels also happens in new-born children, directly after birth, when the portal vein ceases to receive blood from the umbilical veins: jaundice, very frequently presents itself along with defective respiration, especially in children prematurely born, whose fœtal vessels are long in closing up.

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‡ Whether jaundice makes its appearance under such circumstances or not, the secreting function of the gland may become more or less interfered with in consequence.

Moreover, variations of no small importance in the pressure exerted by the hepatic vessels occur, when persistent and profuse hæmorrhages take place from the roots of the portal vein, as in the case of yellow fever.

It is obvious, that the nervous system here, as everywhere else, influences the distribution of blood and its consequences, and, moreover, this has been proved by the experiments of Claude Bernard. We are not, however, in a position to make use of this influence, with any degree of certainty, for the explanation of pathological conditions, because the investigations necessary for this purpose are wanting.* It may suffice, as a preliminary matter, to have briefly alluded here to the importance of the distribution of the blood in the liver, as well as to that of obstruction to the flow of bile in the production of jaundice, reserving the subject for further discussion.

II. *Diminished consumption, or a sparing metamorphosis of the bile which has been taken up into the blood.*

Hitherto, it has been necessary to attribute the tinging of the tissues and secretions, which is observed in jaundice, either to the absorption of bile-pigment, which has been already secreted, or to the hypothetical decomposition of the blood formerly alluded to. There is still another source of bile-pigment in the blood, which is more obvious, of which it may be said that it exists in the blood itself, and which, according to the nature of the transforming causes present in this fluid, must furnish sometimes a greater and sometimes a smaller quantity of pigment, without this being necessarily attributed to an obstruction to the escape of bile.

This view rests upon the following facts:—The pure colourless acids of the bile may be transformed into bile-pigment with all the properties characterising this substance. Such a transformation takes place not only under the influence of re-agents, but it also follows the absorption of the acid substance (into the blood of living animals), and is in a measure dependent upon this.* By the action of concentrated sulphuric acid upon colourless bile, there are

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* From the investigations of Claude Bernard (*Leçons de physiol. expérim.* Paris, 1855, p. 333 *seq.*), it appears to follow, that on the one hand irritating causes, which stimulate certain parts of the nervous system, such as punctures in certain regions of the medulla oblongata, and electric stimulation of the proximal extremities of the divided pneumogastric nerves, and on the other hand, injuries which reduce the nervous energy of the individual, such as contusions of the head, poisoning with curari, ætherisation, &c., give rise to congestion of the liver, whilst division of the spinal cord below the cervical plexus is followed by an opposite result.

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* If concentrated sulphuric acid is poured upon pure, perfectly colourless, glycocholate of soda, there is formed a resinous mass, devoid of colour, which dissolves in the cold with a saffron-yellow colour, and with a reddish colour upon the application of heat. This solution separates into a colourless water, and flakes of a greenish or brownish colour, according to the temperature at which the solution has been made. Glycocholic acid, when changed by sulphuric acid, has the property, upon exposure to the atmosphere, of rapidly taking up acid substances, and of passing into gorgeously-coloured combinations. If the amorphous, colourless mass resulting from the action of sulphuric acid, after it has been deprived, as far as possible, of the adherent acid, is placed upon a piece of filtering paper, it dissolves, and there is produced a ruby-red spot, which soon presents a blue margin, and after a short time assumes an indigo-blue colour. After some days, this colour also disappears, and the spot becomes brown.

By the continued action of sulphuric acid upon glycocholic acid, a substance is produced, which dissolves in water with a deep green colour, and in a weak solution of soda with a brown colour, and which, upon the addition of nitric acid, assumes first a green, then a reddish, and lastly, a yellow tint. The behaviour of this substance with nitric acid reminds us of that which characterises the natural bile-pigment, although the change of colour is less rapid. When taurocholate of soda is treated in the above manner, there is obtained in its place a product behaving in every respect the same as cholepyrrhin. When dissolved in a little water, and mixed with concentrated sulphuric acid, this assumes a brilliant red colour, and gradually, upon exposure to the air becomes blue. When the red solution is mixed with more sulphuric acid, the colour passes into brown. Upon the addition of water, there is produced a delicate precipitate, gradually becoming pale green; if the acid fluid is poured off from this, and what remains is warmed, intense green, blue, and violet colours are produced. The coloured products dissolve in potash, with a bilious brown colour, and the solution behaves, with nitric acid, in precisely the same manner as a basic solution of cholepyrrhin.

That the same metamorphoses may take place in the blood of a living individual is proved by injections of colourless solutions of bile into the veins of dogs. The urine passed after such an experiment usually deposits, upon standing, green flakes, which, upon the addition of nitric acid, exhibit in a beautiful manner the alternation of green, blue, violet, and red colours, characteristic of bile-pigment. The unchanged acids of the bile may then be sought for in vain by means of Pettenkofer's test. In formed colour-producing substances (*Chromogene*),* which, upon exposure to the atmosphere, and still more rapidly on the addition of nitric acid, exhibit alternations of tints corresponding, in every respect with bile-pigment. The same pigments and colour-producing substances (*Chromogene*), which in their properties precisely resemble cholepyrrhin, are produced by the injection of large quantities of colourless bile into the vascular system of living animals. In this case the acids of the bile are transformed in the blood into pigment under the influence of respiration. That the bile which has been re-absorbed from the intestine, or which has passed directly from the liver into the blood, may, under normal circumstances, experience a similar transformation, is an opinion which is favoured in the first place by the presence of large quantities of taurine in the healthy lung, as shown by Staedeler and Cloëtta. The pigments, however, which are produced in this way, are not voided with the urine, until the constantly advancing process of transformation to which the colouring-matter is subjected, has gone so far, that the substance is no longer endowed with the properties of bile-pigment. We not unfrequently find such advanced stages of transformation in the slighter forms of jaundice, sometimes, also, in other forms of disease, in which there is no yellow tinge of the skin to indicate an affection of the liver. Thus, I have observed in a case of slight jaundice resulting from intermittent-fever, urine passed repeatedly of a ruby-red colour, which, on the addition of nitric acid, became blood-red, and remained so for a day,† whilst serous effusions of the ordinary bilious brown colour were found in the dead body. Not unfrequently, the urine which is voided in chronic affections of the

one case only, where an unusually large quantity (about two drachms of dry bile), was injected, a trace of it could be detected. It is worthy of notice, that the quantity of colouring-matter voided in the urine appears greatest, when the animal experimented upon has suffered from dyspnœa, as, for instance, in one dog, which died from œdema of the lung, consequent upon the experiment. In one case, where the quantity of bile injected was small, and the animal remained free from respiratory ailments, no pigment was found at all.

* Chromogen is a term applied by Frerichs to a colourless material, which, when subjected to the action of certain agencies above-mentioned, is transformed into the colouring-matter of bile. The relations of the two substances are somewhat analogous to those of colourless and blue indigo.—TRANSL.

† A similar colouring-matter may be produced by the action of sulphuric acid upon tyrosine.

liver, becomes violet or dark blue on the addition of muriatic acid.* In other cases of jaundice, the urine is brown; but when treated with nitric acid exhibits the play of colours either incompletely or not at all. The bright-red urinary sediments occurring in diseases of the liver have long been familiar to practitioners.

There may thus be detected in the urine very various shades of the same pigment, and it might be difficult, in this intimately-connected series, to distinguish between bile-pigment and the colouring-matter of the urine. The quantity and the quality of this colouring-matter are always influenced in an unmistakable manner by the agencies which modify the metamorphosis of matter throughout the system, and by local diseases which circumscribe respiratory action. In hot summer weather, the urine of healthy individuals often contains distinct evidences of bile-pigment,—a fact which was mentioned long ago by Scherer, and has been repeatedly confirmed by Valentin. A slight and scarcely observable degree of jaundice increases in intensity so soon as any febrile process is lighted up, which restricts, to a considerable extent, the process of oxydation in the blood; and still more remarkable is the influence of pneumonia. These facts, suggest the supposition, that under certain pathological conditions, the metamorphosis of bile in the blood is incomplete, in the same way as is the case after the injection into this fluid of larger quantities of bile, and that thus a sufficient quantity of bile-pigment remains in the blood to give rise to all the symptoms of jaundice. A powerful influence over the quantity of this pigment is exercised by the secretion of urine, which removes it more or less quickly, and thus regulates its accumulation in the blood. (See *Observation No. VII.*) There is, in many respects, an analogy between diabetes mellitus and several forms of icterus; in the same way that in certain states of the system, with which we are still imperfectly acquainted, the sugar which is formed in the liver, in place of being consumed, is voided with the urine, so, under certain conditions, the transformation of the acids of the bile remains incomplete. So far as clinical experience teaches us, this imperfect metamorphosis of bile takes place principally in those diseases which obstruct or interfere with the processes of metamorphosis or oxydation in the blood, such as putrid infection, pyæmia, and its allied conditions, the state of intoxication produced by the bite of serpents, by chloroform, &c., and also the obstructions to respiration from pneumonia, &c.*

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* See Observations on the relations of blue colouring-matter to Bile and Tyrosine, &c. *Müller's Archiv*, 1856.

Thus, to sum up, we have become acquainted with three causes of icterus:—

1. Obstruction to the escape of bile.
2. Diminished circulation of blood in the liver, and consequent abnormal diffusion.

Both of these conditions give rise to an increased imbibition of bile into the blood; and, in both cases, the liver is more or less directly implicated.

3. Obstructed metamorphosis, or a diminished consumption of bile in the blood.

This cause is independent of the liver, and, so far as we as yet understand the matter, is chiefly influenced by the composition of the blood, and by everything which essentially limits or modifies the processes of metamorphosis within the vascular system.

3. *Symptoms of Jaundice.*

We select as the basis of our description of jaundice, that variety which owes its origin to a mechanical impediment to the excretion of bile. This variety is the more eligible, as in it the conditions take on their simplest form and are less apt to be interfered with by disturbances of an extraneous nature, which owe their origin either to the primary cause of disease, or to some other morbid processes going on at the same time.

The bile which stagnates in the hepatic cells and bile ducts, in consequence of a mechanical impediment to its excretion, is carried into the blood by means of the veins and lymphatics. When the impediment has its seat in the ductus choledochus, the bile first shows itself after three days by a yellow colour of the conjunctiva.†

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* In an analogous manner it appears, that the excretion of sugar in the urine, after puncture of the fourth ventricle of the brain, must be viewed in connection with the obstructions to respiration and to the heart's action which result from this wound. (*Bèrlin, Schrader, Uhle, Stockvis.*) Reynoso found sugar in the urine of persons who had been ætherised; Bence Jones in the urine of those who had taken chloroform; and Garrod in the urine of acute bronchitis. It is obvious, however, that here there are other collateral circumstances of an unknown nature in operation.

† Tiedemann and Gmelin (*On Digestion*, Vol. II., p. 48), as also Blondlot, observed jaundice commence after three days, and my own investigations

Its absorption into the blood takes place much earlier;* but in order that the jaundiced colour of the skin should become visible, a certain amount of concentration is necessary. The colour makes its appearance in the renal secretion sooner than it does in the skin, and serous effusions in the various cavities of the body become tinged with pigment earlier than the urine. I have repeatedly found cholepyrrhin in the serum of the blood, and in effusions into the abdominal and thoracic cavities, where no traces of a jaundiced tint had been present either in the urine or in the skin.†

Apart from the tinging of its plasma, the blood undergoes no remarkable change from the absorption of bile; its proximate principles vary, as numerous analyses have shown, according

have usually led me to the same result. After the application of a ligature to the common bile duct, the yellow colour of the conjunctiva was first observed between the sixtieth and seventieth hour, and sometimes later.

* As regards the rapidity of absorption, reference is generally made to the observations of Saunders—(*Abhandl. über die Structur, &c., der Leber. Aus dem Englischen.* Leipzig, 1795, S. 61)—who, so soon as two hours after the application of a ligature to the ductus choledochus, found the lymphatics, as far as the thoracic duct, filled with a yellow fluid, and, in the same space of time, the serum of the blood of the hepatic vein darkly tinged, but that of the jugular vein scarcely at all so. I have not, from my own investigations, been able to confirm these statements. Four-and-twenty hours after the ligature of the ductus choledochus, bile-pigment could not be detected either in the serum of the blood, the lymphatics, or the urine. On one ocasion, it was found in the blood of the jugular vein after twenty-eight hours; but on another occasion, there was not a trace. After forty-eight hours, the colouring-matter could be seen almost always in the blood and in the urine, but not, however, in the contents of the thoracic duct. Sixty hours after the ligature, the chyle was still white on one occasion, but on another it was yellow, and contained pigment. Cases have occurred in which, notwithstanding the complete obstruction of the bile ducts, not a trace of colouring-matter had appeared either in the blood or urine at the end of two days, and others, in which the serum was pale, and without the reaction of bile, but in which the blood, when digested in alcohol for some time, yielded a not inconsiderable quantity of cholepyrrhin, which was first formed from chromogen during standing.

† There are, however, exceptions to this; sometimes, along with the yellow colour of the skin, the urine contains no bile-pigment of the usual reaction, but colouring-matter of another nature, which is formed from it. Moreover, upon *post-mortem* examination, the comparative deposit of pigment is found to be greatest, at one time in the skin and at another in the kidneys.

to the constitution of the individual, &c., without presenting any constant abnormality. Many incorrect statements have been made as to the liquefaction of the blood corpuscles by means of bile; the contents of the gall-bladder possess this property in a less degree than distilled water. Owing to the comparatively small quantity of bile which passes into the blood, for the purpose of undergoing metamorphosis, no liquefying results could be expected from it, although the pure salts of the biliary acids, according to the experiments of von Dusch, are endowed with this property.* It is therefore intelligible how Bécquerel and Rodier† should sometimes have observed even an increase of the red corpuscles: a diminution of the fibrine is, likewise, no proof of the blood having become dissolved.

Of the various elements of the bile taken up into the blood, the colouring-matter is the only one which can always be detected; the biliary acids are apparently changed immediately by the influence of the acid ingredients of the blood, and soon disappear, without leaving a single trace, in the same way that the biliary acids disappear, which, during digestion, are absorbed from the intestinal canal, and return into the circulation. The numerous investigations which have been undertaken since the time of Thénard, to demonstrate their presence in jaundiced blood, have almost always led to negative results.

I have myself repeatedly examined jaundiced blood, which has been obtained by venesection, or still more frequently, from the heart and venæ cavæ of the dead body, for the biliary acids, and their immediate derivatives; and more recently I have had it examined by my assistant, Dr. Valentin, but always with negative results. No substances could be found in the alcoholic extract of the blood which yielded any indication, by Pettenkofer's test for the biliary acids, whether this alcoholic extract was directly treated with sulphuric acid and sugar, or whether, in order to get rid of foreign substances, a watery extract of it was first prepared. This coincides with the experience of most of the older and modern observers.‡

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* It is worthy of notice, that animals, after the injection of bile into the blood, generally pass urine which contains albumen, and the dissolved red matter of the blood. The admixture of these substances, however, soon disappears.

† Untersuch. S. 15.

‡ Thénard, Chevreul, Boudet, Lecann, Deyeux, Gmelin, &c., have

Thus the biliary acids disappear completely from the blood in a short space of time, not because they are voided by the excreting organs, but because they undergo changes, by which they entirely lose their properties.

A long series of fruitless attempts to find these substances, or their immediate derivatives in the urine, the sweat, and the saliva, shows that they are not removed from the blood of jaundiced persons by any increased action of the glands forming these secretions.[*]

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searched in vain for the ingredients of bile. The statements of Orfila, Collard de Martigny, and Clarion, to the effect that they had found the resinous constituent of bile in the blood, were made at a time when it was impossible to distinguish with accuracy between the resin of bile and other similar substances. Since we have obtained in Pettenkofer's test, an easy, although, under certain circumstances, deceitful test for small quantities of biliary acid, it has frequently been resorted to, but in general without results (*Scherer*, V. *Gorup-Besanez*, *&c.*). Lehmann alone mentions his having found a small quantity of the acids of the bile in the blood, in the urine, and in exudations. It appears, however, that this was not in jaundice, but in other diseases in which the liver was not at all implicated. Without wishing to call this fact in question, I must observe, that I have often obtained Pettenkofer's colour in serous and inflammatory exudations in the abdominal and thoracic cavities, and for some time I zealously followed up this observation, until I became convinced that the pure white of egg, when treated with sulphuric acid, often assumes this same violet colour, and that the same exudations which, when employed directly, had yielded a positive result, when dried and extracted with alcohol, ceased to exhibit that reaction.

[*] I have made many attempts to find the materials of bile in jaundiced urine; and, to make the matter more certain, I have operated with large quantities of urine. In order to separate the biliary acids, the urine has been mixed with neutral and basic acetate of lead, and the precipitates have been collected, dried, and then extracted with boiling alcohol. The alcoholic solutions must have contained compounds of lead and the biliary acids, if any of the latter had been present. They were, however, scarcely coloured by the solution of sulphuretted hydrogen, and upon evaporation only a small residue remained, which, with Pettenkofer's test, afforded no indication of biliary acid.

I have met with no better success in endeavouring to extract, by means of alcohol, from the evaporated urine, a substance exhibiting the reaction of the biliary acids.

Upon adding alcohol to the condensed urine, crystals have sometimes separated, which, in their form and in their relation towards solvents, have resembled taurine. The crystals, however, have not been sufficiently large to permit of their angles being measured without a special apparatus. Glycine also has been sought for without success.

In accordance with these results, Griffith, Scherer, Gorup-Besanez, &c.,

Even when we inject large quantities of bile, so as to obviate the difficulties which there might be in detecting a small quantity, we are equally unsuccessful; it was always the colouring-matter only which could be observed. We have, hitherto, been unable to trace in detail wherein consist the changes in the absorbed bile, the occurrence of which we are thus obliged to admit; this much only is certain, that they give rise to colour-producing substances, which, under the action of the (respiratory acid) carbonic acid of respiration, are transformed into bile-pigment. This is confirmed, not only by the quantity of the colouring-matter, which, so far as this can be ascertained, stands in no relation to the quantity daily formed in the liver, but also by the already-mentioned results of injecting biliary acids into the blood, and above all, by the direct examination of the blood of jaundiced patients. We find in it, besides the colouring-matter of bile, colour-producing substances (*Chromogene Substanzen*) which exhibit the same characters as the similar bodies prepared artificially from the bile, and which, like them, become, upon exposure to the air, blue, green, red, and brown.*

The bile-pigment itself, which is obtained by the action of alcohol upon the dried blood, is sometimes amorphous, but at other times it separates in a crystalline form. These crystals consist of short rods, which adhere in rows to one another, and sometimes form radiated crystalline masses,† or appear as angular granules, either

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have likewise never succeeded in finding the biliary acids in jaundiced urine. The assertions of Fourcroy and Vauquelin, that such urine betrays the presence of bile from its bitter taste, are of no importance; neither need much weight be attached to the statement of Orfila, that jaundiced urine contains picromel; or to that of Simon, who believed that he had found in it much biliary resin; because the nature of these bodies has not been established with sufficient accuracy. There appears to be much more importance in the remark of Lehmann, that sometimes a large quantity of biliary acid is to be found in urine, but feebly coloured with pigment, whilst only traces of it occur when the quantity of colouring-matter is abundant. In place of the expected biliary substances, I have always found the pigment only, and usually, in addition, a small quantity of leucine.

* The colour-producing substances were detected in largest quantity in the alcoholic extract of the blood, in those cases where, after the injection of bile, they also appeared in the urine, and likewise in animals two days (*see Note*, p. 94) after the application of a ligature to the ductus choledochus: in other words, under circumstances in which an incomplete metamorphosis might have been expected.

† These are figured in Table VII., Fig. 7, of the Atlas.—TRANSL.

isolated or adhering in groups. They are not very durable, and soon lose their properties; they cannot be re-crystallized; their solution is of a dark colour; and the pigment separates from the solution as an amorphous mass.*

In addition to these colouring-substances, leucine occurs in the blood in considerable quantity,† and there is also an unusual proportion of fat, rich in cholestearine; this last, in some cases, amounts to four or five per cent.‡

Thus the bile-pigment alone remains as the essential substratum of the bilious dyscrasia, resulting from the retention of the hepatic secretion, and to this perhaps may be added, the effects which the metamorphsis of the absorbed biliary acids produces upon the other functions of the blood. It might have been expected, *à priori*, that the consequences of such a change in the composition of the blood upon the vital functions should not be of a very remarkable nature, and in fact, they are not. The violent disturbances which have been attributed to cholæmia, are not due to this, but to entirely different circumstances, which will be discussed hereafter.

The most remarkable effect is the tinging with pigment of the various tissues of the body, and of the individual secretions, which is the more striking the more deeply the blood-plasma is coloured, and the longer this condition continues.

When an obstruction to the excretion of the bile is the cause of the jaundice, the deposit of pigment commences in the hepatic cells. A finely granular, brown or yellow colouring-matter, is first deposited around the nucleus, or the entire cell becomes filled with pale-yellow contents. The nucleus itself remains pale, or it becomes greenish-yellow, or sometimes even dark-brown. At a later period, solid deposits of pigment are observed in the form of small rods, either straight or bulging at their extremities, or repeatedly ramified. Here and there also, the pigment appears as rounded globules, or sharply-angular particles, which have a yellow, reddish-brown, pale, or deep-green colour, and which are hard and may be broken up by means of pressure.

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* The crystalline colouring-matter is insoluble in ether, but is soluble in alcohol; on the addition of nitric acid it exhibits no change of colour; potash dissolves it with a greenish-brown colour.

† In one case, Staedeler also obtained traces of tyrosine in the blood which had been removed by cupping-glasses from a jaundiced patient.

‡ The largest quantity was found in cases of jaundice arising from cancer of the liver, in which it amounted to 3.78 and 4.98 per cent. of the dried blood; in pneumonia with jaundice, there was 1.04; in pyæmic jaundice, 1.97; in typhus with jaundice, 1.93; in cirrhosis of the liver with jaundice, 1.10; and in catarrhal jaundice, 0.90 per cent. It is doubtful whether the increase of fat depends upon the jaundice, or upon the primary affection of the liver: the latter view is the more probable. In cancer of the liver without jaundice, the fat amounted to 3.96 per cent., and was thus more than in simple jaundice.

It is worth observing, that the cells which contain an abundance of pigment are always situated in greatest quantity around the central vein of the lobule, and gradually become less numerous towards the periphery. The co-existence of fat in the cells containing pigment seems to be of rare occurrence, because the cells containing fatty matter have a predilection for the periphery of the lobule.

The cut surface of the liver assumes, in consequence of the stoppage of bile, a more or less intense brown, or sometimes a brownish or olive-green colour. The central portions of the lobules are more deeply impregnated with the colour, which gradually loses its intensity towards their circumference; for this reason, the colour is never uniform, but speckled like a nutmeg. If the obstruction lasts a long time, the bile ducts, and under certain circumstances, the hepatic tissue also, exhibit changes of structure of a more intimate nature, with which we shall subsequently become acquainted, when we speak of the results of jaundice.

Next to the liver and the blood, the jaundiced tint is earliest observed in the serous exudations, and then in the secretions, especially those of the kidneys and of the skin; the colour does not manifest itself everywhere throughout the tissues, to which the blood-plasma is carried, until the accumulation of the dissolved bile-pigment in the blood becomes considerable.

The urine becomes changed in colour at an early period, from the admixture of smaller or larger quantities of cholepyrrhin. It becomes saffron-yellow, reddish-brown, dark-brown, greenish-brown, or brownish-black, according to the quantity and the quality of the pigment which enters into it. A practised eye is usually able, without further tests, to recognise the presence of the colouring-matter of the bile from the saffron-yellow tint of the froth, or of thin layers of the urine; yet this is by no means invariably possible. The urine is not unfrequently of a reddish-brown, or of a brownish-black colour, even when it does not contain a trace of cholepyrrhin. Dark shades of colour, similar to those of jaundiced urine, may be observed in the course of other diseases, especially in impeded respiration, when this is not accompanied with impoverished blood, in emphysema, in congestion of the lungs resulting from disease of the mitral valves, and also in hæmorrhages of the kidney; the urine may also assume a bright-red or saffron-yellow colour after the use of preparations of rhubarb, or santonine, or even independently of these.

Re-agents are almost always necessary, in order to determine with accuracy the presence in the urine of the colouring-matter of bile; and we possess unequivocal tests of its existence. The best we can employ for the purpose is nitric acid, which is not altogether free from nitrous acid. This produces the well-known play of colours from brown to green, blue, violet, and red, this last ultimately passing into a dirty-yellow. This change is exhibited most distinctly by adding the concentrated acid, drop by drop, to the urine in a small conical glass, taking care not to shake it; the different colours are then arranged in layers, one above another like a rainbow. It is not always, however, that this reaction can be recognised in the urine containing bile-pigment. It very frequently happens that the pigment undergoes further changes, either before leaving the blood or in the urine, which deprive it of this property. Even the gaseous acids contained in the atmosphere, may produce similar changes in the urine, when it is allowed to stand exposed to the air; the colour, which at first was brown, gradually becomes greenish, and at the same time the characteristic effect of nitric acid gradually ceases. The modification of the bile-pigment which is produced under such circumstances, assumes, when acted on by acids, a green, or bluish-green colour, which is particularly distinct, when the urine at the same time contains albumen; the precipitate formed by nitric acid then exhibits a bluish-green shade.

However, this reaction may also fail; the colouring-matter may already have passed through all these stages of transformation, and be no longer in a condition to furnish either the one or the other reaction, although, as is proved by the other symptoms of jaundice, it must be regarded as a direct derivative of the bile-pigment. When this is the case, the urine is at one time of a brown, or brownish-red colour, and becomes red on the addition of nitric acid; at another time it is of a deep-red, which is converted, by nitric acid, into a dark bluish-red.

In some cases, the urine contains colour-producing substances (*Chromogene*), and then it exhibits no trace of the characteristic reaction of cholepyrrhin immediately after being passed, which, however, becomes apparent when the urine has stood for some time exposed to the air, and is then treated with nitric acid. It is the same colour-producing substance which is found in the blood of jaundiced patients, and which is formed during the metamorphosis of the acids of the bile into pigment.

The vegetable colouring-matters, which may be mistaken for the pigments of bile, may easily be distinguished by their different behaviour towards re-agents. The colouring-matter of rhubarb and of santonine, which especially come under our notice, become of a blood-red colour when treated with the caustic alkalies or their carbonates, a property which is participated in by no modification of bile-pigment.

Jaundiced urine is usually clear; but in most cases of febrile jaundice, it deposits sediments of uric acid, which are characterised by a bright brick- or rose-red colour. Occasionally, we find precipitates of another nature, such as the epithelium of the urinary passages and of the kidneys tinged yellow; still more rarely, and only in aggravated cases of jaundice, we find flakes of a yellowish-brown fibrinous substance, or of a crumbling, brownish-black pigment, which is sometimes deposited in large quantities in the interior of the tubuli-uriniferi of the kidneys.

A complete examination of jaundiced urine, in reference to the quantity of solids, uric acid, salts, &c., which it contains, has not yet been accomplished on such a scale as to furnish a comprehensive view of the metamorphosis which takes place in the course of jaundice. We are equally ignorant of the amount of the products of respiration. It is not improbable that such an investigation would furnish results, which would repay one's trouble.

By far the largest quantity of the bile-pigment is excreted by the kidneys; the excretion from these organs takes place in such quantities, that in some cases, their texture is essentially injured thereby. In the more persistent and intense forms of jaundice, changes take place in the kidneys, which hitherto have not received the attention which they deserve. The organs assume an olive-green appearance; a number of coiled-up uriniferous tubes of a dark colour may be seen in the cortical substance; and in the pyramidal portion, in addition to brown or sap-green tubuli, there are others which are filled with a black deposit. Upon closer inspection, the paler uriniferous tubules are found to be of a green or brown colour, and their epithelial lining, which is seldom entire, is of a deep-brown tinge, which is particularly marked in the nuclei. The individual cells appear partly of a blood-red colour, partly green, and partly brown; some of them contain layers of pigment deposited in a concentric manner around the nucleus; not unfrequently, we meet with epithelial cells which have undergone fatty degeneration, and are red, brown or black. Where the deposit of pigment is most intense, uriniferous tubes may be observed distended with a coal-black, hard, brittle mass, which, like the material of black gall-stones, is either dissolved in caustic potash, slowly and incompletely, or is quite insoluble. In addition, there may be noticed cylindrical masses, consisting of an amorphous material, brown in the centre, but becoming gradually paler towards the periphery. Caustic potash acts upon them more rapidly, dissolving their pigment, and causing the cylinders themselves to become transparent and swell up like coagulated fibrine which has been retained for a long period in the uriniferous tubes.*

The deposit of pigment under such circumstances extends throughout the entire tissue of the kidneys; it is even perceptible in the epithelium of the Malpighian bodies; it is still more evident in the convoluted tubuli uriniferi, and is pre-eminently distinct in the straight tubules of the pyramids, the calibre of which becomes blocked-up with hard, coal-like masses. It might be imagined, that such a deposit would greatly interfere with the secreting function of the kidneys, and observation proves that it really does so. (See Observation No. VI.)

Next to the kidneys, the sweat-glands take the most prominent part in the excretion of bile-pigment. Not unfrequently, the secretion of the axilla, and of other parts of the skin remarkable for their secreting properties, colours the white linen distinctly yellow. Chomel was acquainted with this symptom,† and Cheyne ‡ observed a patient who first became cognizant of his complaint from noticing his towel become yellow upon wiping his face. Andral § describes a case in which the sweat coloured the linen yellow, and the urine contained the colouring-matter of bile, although the skin and conjunctivæ exhibited no jaundiced tint.

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* These various forms of the pigment-deposit are accurately delineated in the coloured Atlas. Plate I. (Figs. 9, 10, 11, 12).—TRANSL.

† Académ. des Scienc., 1737, p. 69.

‡ Dublin Hospital Reports, Vol. III., p. 269.

§ Clinique Méd., Tom. II., p. 373.

The quantity of colouring-matter which is excreted by the sweat-glands is always very inconsiderable in comparison with that excreted by the kidneys. The sweat-glands themselves do not undergo any important changes of structure, corresponding to those of the kidneys. The contents of the cutaneous glands appear of a somewhat yellow tinge, and here and there may be seen brown granules, and dark nuclear formations, but nowhere a considerable deposit of pigment.

The participation by the remaining secreting organs in the removal of the colouring-matter is very insignificant and inconstant. Wright maintained that he had found cholepyrrhin in the saliva, which, after repeated examinations, I have failed in doing; even in the parenchyma of the parotid and submaxillary glands, and of the pancreas, I have only observed a very slight deposit of pigment.* I have been equally unsuccessful in ever detecting the colouring-matter of bile in mucus. The catarrhal sputa of a woman labouring under very intense jaundice, which was expectorated in large quantity, exhibited the usual greyish-yellow colour, and gave no reaction on the addition of nitric acid (see Observation No. VII.); even the mucus which lined the bile ducts in one case was as clear as water, and without a trace of pigment. (Observation No. VI.) The mucous secretion of the small and large intestines likewise remains greyish-white, and free from colouring-matter; in one case only, have I found it to exhibit a bluish colour, which, however, did not proceed from bile-pigment, but from melanotic granules deposited in the cast-off epithelium. I cannot, therefore, agree with those who, like Fourcroy and others, assume that the mucus is coloured yellow in jaundice. Albuminous and fibrinous exudations are entirely different in this respect: they always abound in colouring-matter. The sputa in bilious pneumonia have a brown, or usually, a leek-green colour, and, upon the addition of nitric acid, present a lively reaction. This property they retain so long as any exudation is expectorated; hence, they are sometimes observed to be still green, long after the jaundiced colour of the skin and urine has disappeared.

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* Huxham (*Opera physico-medica*, Tom. III., p. 12) describes the case of a man, aged 40, who, when suffering from jaundice, and colic from a biliary calculus (*Steinkolik*), became salivated after taking eight grains of calomel, and poured out an enormous quantity of saliva, at first green, and afterwards yellow; the fauces and the teeth were seen to be covered with a substance resembling verdigris. Mercurial salivation, however, causes a transudation of albumen into the saliva, so that the case proves nothing so far as the normal secretion of the parotid is concerned.

Observation No. IV.

Pneumonia Duplex, Icterus, Bilious Stools—Green Expectoration continuing for ten days after the cessation of the Pneumonia, and for eight days after the disappearance of the Jaundice from the skin.

Carl Jänsch, a daily labourer, 64 years of age, was admitted on the 7th of December, 1855. On the 3rd, the patient was carrying a heavy load of wood, which, on going up-stairs, he knocked against the ceiling, so as to produce a violent concussion of the thorax. On the 4th, he had pain in the chest, cough, and bloody expectoration, accompanied with rigors, &c.

On the 11th, the patient was admitted into the Clinique. Pulse, 118; jaundiced colour of skin and conjunctivæ; urine, brownish-black; sputa tenacious, dark-green, and yielding the reaction of bile-pigment. On the right side of the back, there was dulness, with bronchial breathing extending as high as the middle of the scapula; the same signs were observed on the left side, but extending in an upward direction, one intercostal space less; the upper lobes of both lungs normal; stools thin, and coloured with bile. Was ordered infusion of digitalis with gum arabic.

On the 12th, the upper lobe of the right lung was also condensed. The situation and volume of the liver were normal.

On the afternoon of the 13th, the fever ceased; on the 14th, perspiration; pulse, 88. Was ordered infusion of ipecacuanha.

On the 16th, the jaundice of the skin was no longer visible, the condensation of the lungs was receding, and over the dull spaces there could be heard loud consonant rattles.

From the 18th, the urine was free from bile-pigment, but the sputa continued of a dark-green colour, and yielded, with nitric acid, distinct indications of the presence of bile-pigment, until ten days after the cessation of the pneumonia, and eight days after the disappearance of the jaundice from the skin.

Hence it would appear, that in bilious pneumonia the removal of the exudation by means of the expectoration is peculiarly apt to continue. The case also shows, that the expectoration occurs in much greater abundance, and lasts longer than is generally believed.

If now and then mucous fluids have contained bile-pigment, this has always been when albumen has been present, and when, consequently, the mucus has been mixed with exudation.

The statement, also, of Heberden, to the effect that the tears are coloured yellow, I have not been able to confirm.

Cases, however, undoubtedly occur, in which the colouring-matter may be observed in the milk of suckling women. This fact was long since mentioned by Mende and J. P. Frank. Marsh drew off from the distended mammæ of a female who had died of jaundice, a tenacious yellow fluid, which presented all the characters of pure bile; a similar observation was made by Bright;* and more recently, Gorup-Besanez,† has detected unmistakable bile-pigment in the milk of a jaundiced female. This admixture, however, is not constant.

Simultaneously with the tinging with pigment of the secretions just mentioned, changes of colour begin to be observed in the tissues. These are first visible in the skin and conjunctiva.

The skin at first is of a pale sulphur-yellow, and afterwards of a saffron or citron-yellow, or of an olive or bronzed colour (*melas icterus*). The shade of colour depends partly upon the intensity and duration of the disease, and partly upon the smooth or puckered character of the skin, the thickness of its epidermal covering, and the activity of its secreting power. In young persons, whose skin is kept smooth by a thick cushion of fat, we do not find the dark, dirty shades exhibited by the wrinkled skin of older people. The colour is first observed in those places where the epidermis is thin, and the secretion abundant, upon the alæ of the nose, at the angles of the mouth, upon the forehead and neck. Sometimes I have seen the upper half of the body very distinctly tinged without any change being visible on the lower.

Some of the older physicians, such as Morgagni, Behrends, and others, describe a partial jaundice, which in some cases is limited to one-half of the body, and in others is confined to several isolated localities. J. P. Frank, who had an abundant experience in jaundice, never observed cases of this nature. It is probable that pigmentary deposits in the skin of another nature have here been confounded with jaundice; in none of the cases is there any certain proof of the colouring-matter of bile having been present in the urine at the same time.

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* Guy's Hospital Reports, Vol. I.

† Archiv für physiol. Heilk. 1849.

The jaundiced colour of the skin proceeds principally from the deeper layers of the epidermis, the rounded cells of which are coloured intensely, and contain brown molecules; the older flat cells are of a paler tinge. Hence, after the removal of the causes of the jaundice and the disappearance of the colouring-matter from the urine, the colouring of the skin may continue for a long time, until the epidermis is removed by desquamation, and is renewed. This is a circumstance which must not be lost sight of in treatment.

The mucous membranes only exhibit the yellow colour in a slight degree; hence the lips and tongue, especially the latter, when it is covered with a grey coat, present a remarkable contrast to the surrounding saffron-coloured skin.

The yellow colour which penetrates into all the tissues along with the blood-plasma, may be observed in almost all the deeper-lying parts, which can only be examined after death. The adipose cellular tissue, as already pointed out by Valsalva, assumes a citron-yellow colour; in like manner, the serous and fibrous membranes, the areolar tissue, the walls of the blood-vessels and of the lymphatics, and the substance of the bones and of the teeth, become more or less intensely coloured; but cartilage is affected in a less degree. The red colour of the muscles takes on a yellow aspect, owing principally to the tinging of the perimysium* and of the interstitial areolar tissue.

A marked yellow tinge is seldom to be observed in the substance of the brain; I have seen it in a few cases, in which the brain was œdematous, and where the colour proceeded from infiltration of the cerebral substance with yellow serum. The same remark is applicable to the nerves.

In the eye, in addition to the outer membranes, the humours, especially the corpus vitreum, and to a less extent, the aqueous humour, become tinged with cholepyrrhin in the more intense forms of jaundice; † the vitreous body assumes a pale citron-yellow colour, and, on the addition of nitric acid, exhibits distinctly the reaction of cholephyrrhin, as does also the watery fluid contained in it. I have never observed this character in the lens.

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* The perimysium is the areolar sheath, which envelopes the muscles, as well as their larger and smaller fasciculi and individual fibres.—TRANSL.

† It is only in the intense forms of jaundice, that I have been able to detect this change; as a general rule, there has been no abnormal tinging of the humours of the eyeball.

In pregnant women, the fœtus participates in the yellow colour. Bonetus,[*] long ago, thus described a fœtus born of a jaundiced female: "Ita flavum, ut e cera confectus puer non partus humanus videretur." Similar observations were made by Wrisberg and Finke.[†] In order, however, that the offspring should become jaundiced, a long continuance of the disease is necessary; in jaundiced females who have aborted from five to fourteen days after the commencement of the jaundice, I have been unable to perceive any alteration in the colour of the fœtus. (See Observations No. XIII. and No. XIV.)

Along with the alteration of colour, although of less constant occurrence, a series of symptoms are observed which proceed from abnormal conditions of the nervous system. The principal of these are the following:—

1. *Itchiness of the skin,* in many cases, although by no means in all (according to my own experience only in one-fifth), accompanies the commencement of the jaundice. The itchiness is, in most cases, spread generally over the skin, and is especially troublesome in the night-time; in a few cases it is partial, or confined to certain localities of the skin, such as the axilla, the inguinal region, &c.; it usually disappears after some days, or when the jaundice increases in intensity. Graves observed one case, in which the itchiness showed itself before the commencement of the jaundice, on the outbreak of which it disappeared. In most cases it produces no change in the skin; sometimes there appears an eruption of pimples or pustules, which are usually scratched at an early period; and, in exceptional cases, according to the experience of Graves, urticaria may be present. In one person labouring under jaundice, I observed the skin covered with numerous rounded wheals (*Quaddeln*) the size of a four-groschen piece;[‡] these produced no inconvenience, and disappeared eight days after they first showed themselves.

2. A second, though also not an invariably concomitant symptom, is *derangement of the general sensations:* great exhaustion and debility, along with sadness and peevishness of temper, headache, giddiness, &c. These symptoms are often completely wanting in simple jaundice; in other cases, they are sufficiently explained by the coexistence of catarrh of the stomach; in chronic cases, they are

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[*] Sepulchret, T. II., p. 333.

[†] De morbis biliosis anomalis.

[‡] $\frac{9}{10}$ English inch, or about the size of a shilling.—TRANSL.

not unfrequently observed to occur independently of any other source, and then they betoken, for the most part, nervous disorders, which depend not upon the jaundice, but upon other derangements of the functions of the liver, which will hereafter come under consideration. Still rarer are,

3. *Abnormal perceptions of the senses, subjective sensations of taste and sight.* A bitter taste with a clean tongue, of which complaint is now and then made, usually disappears after a few days. That this may depend upon the accumulation of bilious matter in the blood, is proved by the experiment which I have often made of injecting bile into the veins of dogs; as soon as the fluid enters, the animals keep on continuously licking with their tongues. The bitter taste frequently depends upon the eructation of the bilious contents of the stomach.

Another delusion of the senses met with in jaundiced people has at all times attracted much notice, yellow sight or xanthopsy. The ancients appear to have been acquainted with this symptom, if we can rely upon a passage in Lucretius.* Fr. Hofmann † mentions two cases: since then it has been several times, although comparatively seldom, observed. J. P. Frank met with it only in five out of a thousand cases of jaundice; I myself have never had an opportunity of seeing a case, although I have always made inquiries on the point.‡ White objects appear to the patients to be yellow, in most cases, only for some hours, but sometimes for several days. In earlier times, this abnormal condition of the function of vision was considered to be produced by a yellow tinging of the cornea, and of the aqueous humour, which had been observed to accompany this symptom.§ In opposition to this view, however, J. P. Frank brought forward the following considerations: that he had met with a yellow condition of the cornea, without the derangement of vision, just alluded to; secondly, that the yellow vision intermits; and lastly, that the same symptom is met with in typhus fever without jaundice. Hence, in addition to the jaundiced colour of the membranes and fluids of the eye, Frank blamed a morbid action of the nerves. The latter has in recent times been usually regarded as the sole exciting cause; first, because this symptom is frequently absent in intense colouring of the tissues of the eye; and secondly, because other derangements of the faculty of vision, such as day- and night-blindness, present themselves under the same conditions as those which give rise to yellow vision. Stokes considers yellow vision as an indication of impending paralysis, and Bamberger observes, that he has only met with it in jaundiced persons who have died of cirrhosis of the liver. The analogous condition, however, which is observed after the use of santonine appears to me to argue in favour of the importance of the accumulation of colouring-matter in the blood, and in the fluids of the eyeballs. Here also, whilst the santonine is being transformed by the alkalies of the blood into a coloured modification, all objects in a feeble light appear of a greenish-yellow colour, but this coloured vision ceases so soon as the colouring-matter is excreted by the kidneys. Elliotson's case of jaundice, in which the yellow vision was limited to one eye covered with varicose vessels, whilst the other eye saw colours unchanged, might admit of another simpler explanation.

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* "Lurida præterea fiunt quæcunque videntur arquatis."

† Med. rat. Syst., Tom. IV., p. 353.

‡ In interrogating patients concerning this symptom, especially if we have to do with those who are uneducated, we must exercise great caution, if we wish to receive trustworthy answers; I have sometimes received replies in the affirmative, which, upon closer inquiry, have been withdrawn.

§ See Morgagni, *De Sed. et causis morborum*, Epist. 37-8; and J. P. Frank, *De curandis hominum morbis*, lib. VI., pars III., p. 343. Frank's remark that the vitreous humour does not become partly yellow is erroneous; I have always found more colouring-matter in it than in the aqueous humour.

4. *Retardation of the heart's action.* Very commonly the frequency of the heart's contraction in jaundice falls to a greater or less extent below the normal standard, in most cases to 50 or 40 beats, and now and then to still fewer; in one case I have counted 28 beats, and in another only 21. This retardation, which not unfrequently is accompanied by irregular rhythm of the heart's action, often lasts for several weeks before it disappears; it ceases immediately, when inflammatory or other acute processes supervene as complications of the jaundice, and a moderate frequency becomes substituted. When jaundice appears in the course of any febrile disease, such as acute catarrh of the gastro-intestinal canal, typhus fever, &c., there is, in most cases, a remarkable diminution in the frequency of the pulse as soon as the yellow colour shows itself: the pulse sinks from 110 to 80 or 70, or even less.

The slow pulse is no constant symptom of jaundice; cases occur in which it remains absent during the entire course of the disease.

Why this is so, cannot be determined with certainty, any more than we can in general explain the cause of the phenomenon in question. It might be assumed, that the bile exercises an influence upon the vagus nerve, or upon the brain, similar to the action of digitalis; but we do not possess any positive proofs of such an assumption; and the complete integrity of all the remaining functions of the nervous system, as also the condition of respiration, renders it very improbable. Whilst along with the diminished frequency of pulse which follows the use of digitalis, the respiration is wont to become more frequent; in jaundice, on the other hand, the frequency of respiration diminishes along with that of the pulse, although not in the same ratio. The number of respirations in proportion to the beats of the pulse is, in most cases, as 1 to 3. It is conceivable, that the stimulating action of the blood upon the muscular tissue of the heart may be diminished, or that there may be adhesion of the blood to the coats of the vessels, but at present it is impossible to come to any decision upon the matter.

5. *The temperature* in simple jaundice remains unchanged. It varies in the axilla from 36.8° to 37.25° cent. (98.24° to 98.85° Fahr.); such a temperature we find in individuals who have been suffering for a long period from carcinoma of the liver, or from other organic diseases. It is obvious, that the circumstances are different in the cases of febrile jaundice; in catarrhal jaundice with fever, we have found the temperature 38.5°, &c. (101.3° Fahr., &c.)

6. *Derangements of digestion.* The functions of the stomach in jaundiced persons are usually unaffected; the patients may have a clean tongue, and may enjoy an appetite which leaves nothing to be desired. We meet first with abnormal conditions of those processes, which go on in the intestine, and which are known to depend upon the deficiency or absence of bile in that tube.* Observations made by establishing biliary fistulæ, which, in recent times, have so frequently been repeated, have certainly proved, that the absence of the hepatic secretion in the intestine produces no derangements of nutrition by any means remarkable, or threatening life; that, on the contrary, most of the objects of chylification can be attained without the presence of bile; still, however, there arises a series of abnormal

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* Sometimes the appetite is morbidly increased, and there is a craving for peculiar articles of diet, such as shellfish, mussels, &c., as in a case observed by Budd.

conditions, the influence of which gradually increases, and, in jaundice of long-standing, terminates in defective nutrition.

The stoppage to the flow of bile first interferes with the processes of diffusion which take place in the upper part of the intestinal canal, between the fluid portion of the chyme and the blood in the interior of the vessels. It must be of some consequence to these processes, whether or not in the space of twenty-four hours 1 kilogramme (2.2046 lbs. avoir.) of fluid, more or less, is mixed with the intestinal contents.

The want of bile of course does not influence in any remarkable manner the digestion of the albuminous and carbonaceous ingredients of the food; but according to the experiments of all practical inquirers upon this subject, the absorption of fat is considerably restricted. Jaundiced persons have in general an aversion to fatty articles of food, and after eating them a large proportion of the fat appears in the evacuations. The loss in nutrition, which results from this cause, is sufficiently great to become observable in the course of time. Another result of less moment is the loss of the antiseptic influence of the bile, which permits of abnormal transformations of the intestinal contents, and the generation of large quantities of gas. Hence, flatulence is generally complained of by jaundiced patients, especially when they exhibit a preference for animal food; in such cases the fæcal matters emit a putrid odour, they differ greatly from the normal evacuations, and they contain numerous vibriones, and substances which in their chemical characters resemble those which are found in putrefying albumen and caseine. When, on the other hand, the food consists principally of vegetable, amylaceous substances, the evacuations have in general no remarkable odour, and are of an acid nature, because a part of the carbonaceous food undergoes acid fermentation * in its transit through the intestinal canal. This, however, likewise occurs in healthy conditions, so that it might be difficult to determine, whether it is increased in proportion by the removal of the bile.

Of greater importance in a practical sense are the changes in colour which the fæces are wont to exhibit in jaundice, because from their characters we can most easily draw a conclusion as to the more or less complete exclusion of bile from the intestine. When the obstruction of the biliary ducts is complete, every trace of bile-

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* Monro and Pringle long ago remarked the sour smell of the fæcal matters.

pigment disappears from the stools;* they assume an ash- or clay-colour, which only varies according to the nature of the food. Their consistence is almost always at the same time increased; they become hard and firm; the bowels are also sluggish and require stimulation by means of purgatives. This tendency to constipation is so constant in jaundice, that the assumption appears completely justified that it is owing to the want of bile in the bowel. Whether the bile favours evacuation by stimulating the peristaltic motions of the intestine, or by increasing the secretion of the intestinal glands, or by liquefying the ingesta, are questions which we may leave undecided. Spontaneous diarrhœa in jaundice is of rare occurrence; I have observed it repeatedly as a consequence of dysentery, which now and then makes its appearance in the course of the disease. More frequently we find, that notwithstanding the exclusion of the bile from the bowel, the stools come gradually to be passed regularly.† The characters of the fæces are not always such as have just been described. The colour certainly often appears paler than usual, but still the pigment is not entirely wanting. This is always the case, when the exclusion of the bile is incomplete, whether this be owing to only a portion of the biliary ducts being compressed, or because there is merely constriction of the principal duct, which impedes, but does not completely arrest, the passage of the bile. The former condition frequently happens in cirrhosis, where the extreme ramifications of the biliary ducts are partially obliterated by the compression of the newly-developed areolar tissue; also in carcinoma and other tumours which are wont to encroach upon certain of the larger branches only; the latter condition again is observed in catarrh of the ductus choledochus and ductus hepaticus, in which the tumefaction of the mucous membrane entails only an impediment to the flow of bile, in angular concretions, which cannot completely obstruct the flow, &c.

Occasionally we find jaundiced persons passing stools of a normal colour, or unusually dark. There may be two reasons for this. Either the cause of the biliary obstruction has been suddenly removed, and the bile passes again into the bowel, whilst the colour of the skin remains unchanged, as often happens in the case of concretions, and other rapidly-disappearing causes of obstruction; or, there is a sudden cessation of an excessive absorption of bile (*Polycholia*), of which we shall subsequently speak.

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* Osborne (*Dublin Journal*, February, 1853) supposes that the mucous membrane of the intestine can secrete dark bile, like the external integument and the kidneys, and that in this way the fæces may become coloured, notwithstanding the closure of the bile ducts. This, as far as my experience goes, is never the case.

† Graves and Stokes. Dublin Hosp. Reports, Vol. V., p. 109.

4. *Duration of Jaundice.*

The duration of jaundice is very various; it may fluctuate between a few days and several years. The determination of the longer or shorter duration of the disease depends principally upon its primary causes, the persistence of which may vary, and which may of themselves help to bring about a fatal termination speedily or slowly. If we except those forms, in which the other consequences which may result from the primary exciting cause of the jaundice, decide the matter, the jaundice which proceeds from simple obstruction to the flow of bile, as when the ductus choledochus is obliterated, may last for years before it terminates fatally. Graves and Stokes* mention two cases of jaundice, in one of which the disease lasted for eleven months, and in the other for two years, before the nutrition became impaired; Budd† saw a man, who during an attack of jaundice which lasted four years, with complete obstruction of bile, continued well nourished; Deway‡ has described a case of seven years' duration, and Van Swieten § mentions the case of a female who suffered from jaundice for eleven years, and was ultimately cured by solvent extracts.

The cases which I have had an opportunity of observing, have terminated much more rapidly, even when the primary causes of the jaundice have not contributed in any other way to the acceleration of the fatal termination of the disease. Thus, one woman with obliteration of the ductus choledochus died eight months after the commencement of the jaundice; another died after six months and fourteen days; one man with a cancerous tumour of the duodenum, the size of a walnut, only survived the first appearance of jaundice nine weeks. In one case only, in which a biliary concretion had closed up the ductus choledochus, did the jaundice persist for two years and a-quarter.

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* Dublin Hospital Reports, Vol. V., p. 103.

† *Op. cit.* p. 371.

‡ Gazette méd. de Paris. 1843.

§ Comment. III., p. 130.

5. *Modes of Termination of Jaundice.*

The jaundice does not disappear completely until some time after the removal of the causes which have occasioned the accumulation of colouring-matter in the blood. Where the cause of the jaundice consists in an obstruction to the passage of bile into the intestine, the recovery is announced by a return of colour to the stools; their colour becomes darker by degrees when the disappearance of the obstruction is gradual, as in catarrh of the bile ducts; or they are rapidly overcharged with bile, when from the sudden removal of the obstruction, the pent-up secretion of the liver at once finds its way into the bowel, as in the case of concretions, &c. At the same time, the pigment begins to disappear from those parts which, under normal circumstances, are free from it. First, it disappears from the blood and from the urine; the solid tissues remain coloured for a longer period, and particularly those tissues in which the molecular changes take place slowly. The colouring matter deposited in the epidermal layer of the skin disappears gradually as this layer becomes regenerated by desquamation and new formation; hence, it may last for weeks, especially in old persons. The colouring-matter appears to be gradually washed out from the other tissues by the current of the nutritious fluid. Thus the complete removal of the jaundiced colour always occurs much later than the cessation of the disease,—a fact which must not be lost sight of in the application of remedies.

Not unfrequently jaundice terminates in death, which may be brought about in very various ways. Passing over the influences which the manifold exciting causes of jaundice may, directly or indirectly, exercise in this direction (and indeed the mode of fatal termination is as varying as the etiology of the disease, and in most cases death terminates the process sooner or later), we confine ourselves, for the present, to the consideration of those injurious effects by means of which the simple retention of bile may gradually undermine the constitution, and lead to death. The accumulation of bile in the blood is followed by no dangerous consequences; it is only in rare cases that the deposit of pigment in the parenchyma of the kidneys impairs the functions of these organs to an alarming degree.* The danger to be apprehended almost always proceeds from the consecutive changes in the liver itself which result from the retention of bile. The pent-up secretion gradually distends to a greater or less degree, the biliary ducts as far as their finest ramifications; stretching through the parenchyma may be seen the cylindrical, or not unfrequently, ampulliform ducts, which compress the adjacent glandular tissue, and a part of the ultimate ramifications of the portal vein.

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* Deway (*Gaz. méd. de Paris*, 1843) observed complete suppression

FIG. 19.

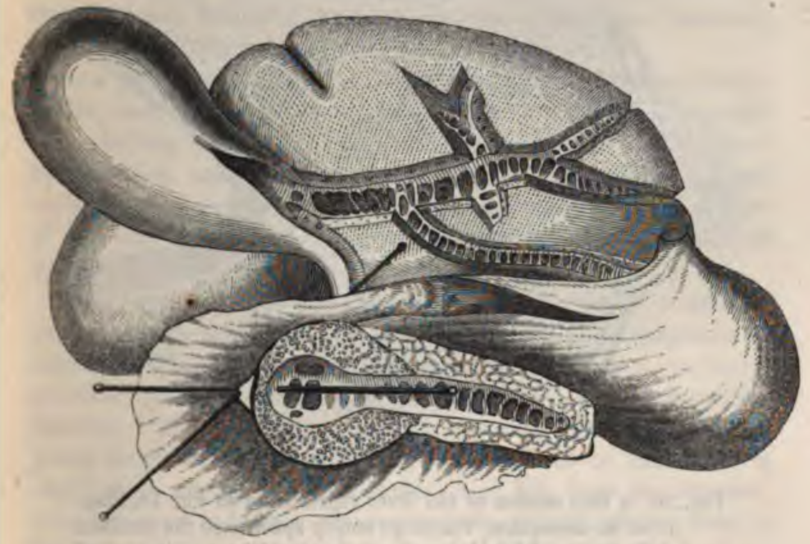

FIG. 19. Enlargement of the bile ducts, and of the pancreatic duct, in consequence of a cancerous tumour in the head of the pancreas. The details of the case from which the preparation was obtained will be afterwards given under Observation No. VII.

(Fig. 19.) When we make a fine section of the hardened substance of such a liver, and examine it with a moderate magnifying power, numerous deficiencies are seen throughout its tissue, usually situated at the periphery, but sometimes near the centre of the lobules, and here and there forming large empty spaces. (Fig. 20.)

The walls of these distended ducts are in most cases considerably thickened. Their contents in general consist of a thin fluid bile,

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of the urinary secretion in a case of intense jaundice, during the last three days of life. See further, Observation No. VI.

which is mixed with a varying quantity of the mucous secretion of the bile ducts. Occasionally it happens, that the bile is thickened and lines the inner surface of the enlarged ducts in the form of a solid, tubular, dark-brown mould. I have seen this in one case, in which there was an exuberant cancerous growth upon the mucous membrane of the bile ducts; and Stoll long ago recorded an instance of a similar nature. Sometimes, notwithstanding intense jaundice of the liver and other tissues, there is not a trace of bile in the fluid distending the gall-bladder and the excretory ducts. This consists of a colourless transparent liquid, containing a small quantity of mucus along with greyish flocculi and mucous corpuscles. (See Observation VI.)

FIG. 20.

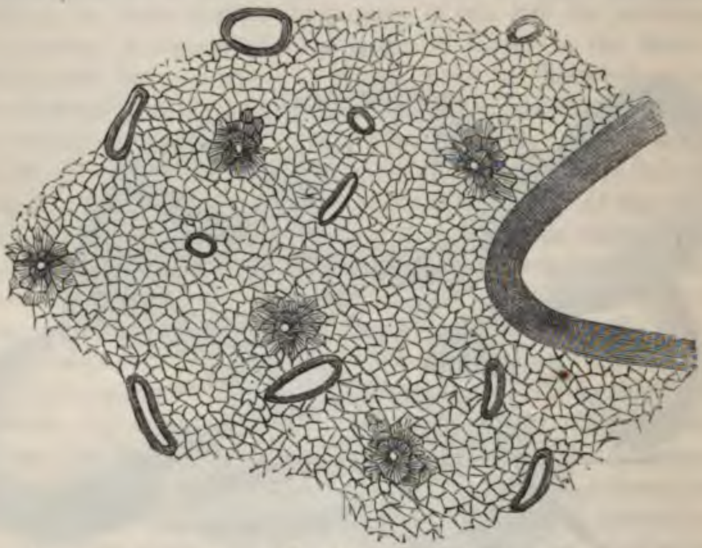

FIG. 20. A thin section of the liver represented in Fig. 19, magnified 80 diameters. The large empty spaces are the sections of the enlarged bile ducts, the walls of which are represented as much thickened. The smaller openings, surrounded by dark spaces, are the sections of the hepatic veins, surrounded by cells loaded with bile-pigment. The shading around these openings should have been of a granular character.

Simultaneously with the distention of the bile ducts, a large proportion of the hepatic parenchyma becomes atrophied and destroyed in the manner just mentioned; and another portion is rendered incapable of performing its function owing to the accumulation of bile within the cells. In this way, the secreting powers of the gland are more and more impaired, and the circulation of the blood in it is obstructed. In some cases, the function of the liver is completely arrested, owing to the glandular cells crumbling down into a finely granular debris, in a similar manner to what is observed in acute atrophy. This last change appears to be brought about by the circulation becoming impeded, and the parenchyma becoming overladen with the products of secretion.

As a general rule, it seldom happens, and that only when foreign bodies, such as concretions, are present, that the gall-bladder or the distended bile ducts on the upper surface of the liver are destroyed by ulceration, and that their contents, passing into the abdominal cavity, excite severe peritonitis. In a similar manner, extravasations of bile may be supposed to take place within the liver, and to lead to the formation of abscesses.

Under such circumstances, the case terminates fatally, sometimes by exhaustion, sometimes, on the other hand, with symptoms of blood-poisoning from the so-called cholæmic intoxication, or, lastly, at other times, by perforation and peritonitis, or by suppurative hepatitis. The first mode of death is the most common. The impaired nutrition, which has been already induced by the defective chylification arising from the exclusion of bile from the intestine, becomes more and more obvious, and the volume of the liver, at first enlarged from the accumulation of the bile, begins to decrease. The organ becomes flabby, shrivelled, and wrinkled, because the absorption of the stagnating bile exceeds the gradually abating secretion.* At the same time, attacks of gastro-intestinal catarrh result from the obstructed circulation of blood, and, in some cases, hæmorrhages take place from the stomach and intestine.† Dropsical effusions form in the peritoneal sac, which are wont to be followed by symptoms of hydræmia and general anasarca. In this way the fatal termination approaches imperceptibly; or, as in other conditions of exhaustion, it may be brought about more speedily by inflammatory exudations, such as pneumonia, pleurisy, peritoneal exudations, dysentery, &c. The second form of death, that from intoxication of the blood, takes place when the function of the liver becomes entirely arrested, owing to the disintegration of the hepatic cells. In such cases the patients are restless, complain of headache, and delirium, and convulsions supervene, which may pass on to coma and death. (See Chap. V., on *Acholia.*) When the bile ducts are ruptured, the train of symptoms characteristic of a rapid peritonitis close the scene; or death may be preceded by the symptoms of hepatitis, passing into suppuration.

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* Budd (*Op. cit.*, p. 198) is of opinion that the diminution of the liver is due to the crumbling down of the hepatic cells; and to prove this, he appeals to an examination made by Williams (*Guy's Hospital Reports*, October, 1843), confirming this disintegration of the cells. This is a mistake; the liver may become remarkably small, without any destruction of its secreting cells.

† Observations of this nature have been recorded by Bright (*Guy's Hosp. Rep.*), Durand, Fardel (*Arch. général.*), Andral (*Clinique méd.*), Budd (*Op. cit.*, p. 203).

6. *Diagnosis.*

The diagnosis of jaundice is in general not difficult, as, usually, simple inspection in daylight is sufficient.* It is only the slighter degrees of the affection which are overlooked or confounded with changes of colour in the skin depending upon some other cause, as, for instance, with the dark colour which results from sun-burning, with the greyish-yellow colour which is observed in the cancerous cachexia, intermittent fever, lead-poisoning, &c., with the yellowish-green tint of several cases of chlorosis, and with the yellow colour which is wont to supervene upon the erythema of new-born children. All these abnormalities of colour are distinguished from jaundice by the conjunctiva† and the urine remaining free from bile-pigment. It must not, however, be forgotten, that in some individuals the membrane covering the eyes has a yellow appearance dependent upon adipose cellular tissue subjacent to it (which, however, may easily be distinguished from the colour of jaundice by its unequal distribution); and it must also be remembered that yellow and brown colouring-matters, bearing a striking resemblance to cholepyrrhin, may be present in the urine. We have already discussed how these sub-

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[*] In artificial light, the jaundiced colour, even when it is intense, is easily overlooked.

[†] There are rare exceptions, in which, notwithstanding the jaundiced colour of the skin and the presence of bilious matter in the urine, the conjunctiva remains unaffected. I have seen two attacks of this nature in a very anæmic individual.

stances may be recognised and distinguished, and how, generally speaking, the urine may be examined so as to detect with certainty any bile-pigment which may be present.

The real difficulties in diagnosis commence when we come to determine the causes of jaundice. We shall endeavour to solve these difficulties, so far as is possible, in describing the various sorts and forms of jaundice; the diagnosis of many of the etiological causes can be treated of subsequently, under the head of the corresponding diseases of the liver.

7. *Prognosis.*

The prognosis, in jaundice, depends principally upon its causes: the natural course and modes of termination of the primary disease, and the greater or less probability there is of interfering with effect in the way of treatment, constitute the considerations which alone will enable us to predict the result. An accurate etiological knowledge of each individual case furnishes us with the prognosis at once; only where this is impossible, are we wont to remain in doubt as to the result. It must not, however, be forgotten, that in apparently simple cases of jaundice, without any organic lesion of the liver, symptoms of blood-poisoning sometimes make their appearance suddenly and unexpectedly, and then, as a general rule, death supervenes in a short space of time. We are not yet in a position, as will subsequently be shown more in detail, to recognise these forms at their commencement, and, for this reason, even in simple jaundice, we can never form a perfectly certain opinion as to the result.

8. *Treatment.*

In the treatment of jaundice, the first point to be considered is the causes which have led to the accumulation of bilious matter in the blood. When these are removed, there is seldom any necessity for further treatment directed against the jaundice itself.

As might be expected, the means to be employed for accomplishing these indications for treatment, vary greatly according to the nature of the primary causes of the disease; they will subsequently occupy our attention, when we come to treat of the individual forms of jaundice and of the corresponding affections of the liver and of the bile ducts.

Not unfrequently, the cause of the jaundice cannot be influenced by treatment; and in such cases, what we have to do is to counteract, in a suitable manner, the injurious influences which the abnormal distribution of bile may exercise upon the entire organism. For this object there are three principal indications which we must keep in view:—

1. The regulation of the functions of the bowels which have become deranged by the stoppage to the flow of bile.

2. The purifying of the blood from the mass of colouring-matter which has accumulated in it.

3. The consideration of the further consequences, which may befall the entire system, from the operation of the above abnormal conditions, and more especially from the changes in the tissue of the liver resulting from the stoppage of bile: such consequences as anæmia, dropsy, cholæmia, &c.

The derangement of the functions of the bowels, which manifest themselves principally in the form of obstinate constipation and complaints of flatulence, may be greatly lessened by means of an appropriate choice of diet, by restriction to easily-digested lean meat and vegetable food, and by avoiding all fatty articles and such as are wont to occasion flatulence. The sluggish condition of the bowels is best stimulated by means of the extract or infusion of rhubarb, or by small doses of aloes, or of elixir proprietatis,* or, if necessary, by tincture of colocynth. The saline purgatives are not suitable for constant use in such cases; they are only adapted for catarrhal jaundice which supervenes upon gastro-intestinal catarrh. When the flatulence is very troublesome, we may infuse along with the rhubarb the root of the calamus aromaticus, peppermint, &c., or we may add some ether to the infusion.

As regards the second indication for treatment, the kidneys contribute more than any other organ to purify the blood from the colouring-matter of the bile; and next to them come the cutaneous glands. The secretion of urine—which, in the later stages of the more intense forms of jaundice, is not unfrequently suppressed to an alarming degree, in consequence of the deposit of colouring-matter in the parenchyma of the kidneys—must be stimulated from time to time by means of diuretics. We employ for this purpose the milder vegetable diuretics, and small doses of the neutral salts, such as borax, tartrate of potass, acetate of potass, &c., and also Seltzer and other mineral waters of a similar nature. Valleix has particularly recommended nitre, in doses of from 4 to 6 grammes (ʒj to ʒjss) in the day. According to my own experience, lemon-juice acts more favourably in doses of 1½ to 3 ounces daily: this agrees best with the digestive organs, and excites an abundant diuresis.

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* The *Elixir proprietatis Paracelsi* is a remedy frequently mentioned in German works on medicine. It is a spirituous solution of aloes, myrrh, and saffron, with the addition of a little dilute sulphuric acid.—TRANSL.

The cutaneous functions may be stimulated by means of tepid baths and mild diaphoretics; baths, to which some ounces of soda are added, are particularly suitable for the removal of the deposit of pigment in the epidermal layer of the skin, when this persists long after the removal of the obstruction in the bile ducts. The functions of the skin are to be kept within due bounds, by stimulating, at the same time, the secretion of urine; and every cause that tends to derange the digestive functions must be avoided.

We experience most difficulty in following out the third therapeutic indication, the counteraction of the injurious consequences to the system, resulting from the consecutive atrophy of the liver, from the destruction of numerous branches of the portal vein by the pressure of the enlarged bile ducts, and lastly, from the disintegration of the hepatic cells.

The cachectic and anæmic condition, which is not noticed until the shrinking of the hepatic parenchyma and the obstruction to the circulation in the portal vein have become considerable, is best treated by means of bitter medicines, which assist digestion, and by a carefully selected, easily assimilated diet, both of which remedial measures are the more necessary, the more that the functions of the stomach are embarrassed by the state of passive congestion produced in it by the obstruction to the flow of blood. Mild preparations of iron, such as the carbonate and lactate, and small doses of mineral waters, such as those of Schwalbach and Pyrmont,* are also sometimes to be tried. In the case of dropsy, which, in general, commences with ascites, we must depend chiefly on the tonic treatment just indicated, alternated with mild diuretics; the use of strong purgatives in such cases is not permissible.

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* The waters of Schwalbach are chalybeate and gaseous, containing carbonate of iron, held in solution by a great excess of carbonic acid. Their temperature is about 50° Fahr. The Springs of Pyrmont are of a similar nature, and had at one time an unrivalled reputation, although of late years they have been much less resorted to.—Transl.

Treatment is of no avail in the state of acholia, which, in some cases, supervenes as a consequence of the consecutive softening of the hepatic tissue; here, a purely symptomatic treatment is all that remains to us.

Empirical Treatment.

A whole series of empirical remedies, which have been tried by various experimenters, have been recommended in the treatment of jaundice. Most of these owe their importance to the relation in which they stand to the causes of the jaundice, although, of course, I do not mean that they have any specific relation to this. Hence, in the choice of these remedies, we can only be guided by the etiology of the disease.

1. First among these remedies, stand *purgatives,* of which at one time the neutral salts, but especially calomel (*Michaelis, Hufeland*), and at other times the vegetable bitter and drastic purgatives, such as rhubarb, aloes (*Pitschaft*), colocynth, &c., have been preferred. These act by stimulating the secreting functions of the intestine, and by exciting their peristaltic motions, which, being transmitted to the bile ducts and the liver, promote the excretion of the bile, and so overcome the slighter forms of obstruction, such as catarrhal tumefaction of the mucous membrane, the smaller concretions, &c. These remedies are to be employed in moderation, and every cause of exhaustion is to be avoided. It is still undecided whether the preference ought to be given to calomel; but at all events it is not advisable to push the use of it as far as salivation. The watery extract of aloes, in doses of from half-a-grain to two grains, or tincture of colocynth, in from five to ten drops, several times in the day, are in most cases sufficient for the object in view.

2. *Emetics* of tartar emetic, or ipecacuanha. Fr. Hoffmann has especially recommended the former, whilst Richter and Baldinger have spoken in favour of the latter. More recently, Corrigan has recommended ipecacuanha to be taken in doses of 2 grammes (ʒss) every second day. Emetics, when employed under such circumstances, are certainly very effectual in overcoming the obstruction to the excretion of bile. During their action, the liver and bile ducts are powerfully compressed on three sides, so that the fluid contents of the latter are pushed with great force against the obstruction. In dogs, after violent vomiting has been excited by the

injection of tartarized antimony into the veins, I have found the bile ducts, for the most part, completely empty. In the case of catarrhal jaundice and in gall-stones, emetics not unfrequently succeed in freeing the bile ducts, although in the latter case their violent action may be dangerous, inasmuch as it may give rise to rupture of the gall-bladder, and escape of bile into the peritoneal cavity. Hence, under such circumstances, they must be used with caution.

3. The *solvent extracts,* such as the extractum graminis, ext. taraxaci; ext. card.; ext. chelidon.,* &c., owe their effects partly to the salts which they contain, and partly to the bitter ingredients, by virtue of which they exercise a favourable influence in chronic catarrh of the gastro-duodenal mucous membrane. Whether the compounds of the alkalies with the vegetable acids contained in these extracts can contribute towards the increase, or to any change in the quality of the biliary secretion, must remain uncertain until unequivocal experiments, by means of biliary fistulæ, clear up the matter.

The ancients attached great importance to these extracts, as also to the freshly-expressed juices of plants. Van Swieten records in detail the cure of a case of very lingering and obstinate jaundice by means of decoction of grass.

4. The *muriate of ammonia* has a similar effect upon gastro-duodenal catarrh, and was especially recommended by Baglivi† in the treatment of jaundice; the same may be said of the purified tartrate of potash and soda (*tartar-depuratus natronat.*), and the allied salts.

5. *Narcotics,* such as cicuta (*Stoerck*), belladonna (*Richter*), theriac‡ (*Galen*), may be employed effectually in those forms of

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* *Extractum Graminis* is the extract of the root of the Triticum repens or Couch Grass, which is said to contain sugar and free oxalic acid.

Extract. Cardui benedicti is an extract of the leaves of the Carduus benedictus, a species of thistle, the chief constituent of which is a bitter extractive matter.

Extract. Chelidon. is the extract prepared from the juice of the Chelidonium majus or celandine. It is acrid and bitter, and sometimes is followed by narcotic effects.—TRANSL.

† Prax. medic., lib. I., de ictero flavo.

‡ *Theriac,* is the *Electuarium Theriaca, seu opiatum, seu aromatico-opiatum* of the Prussian and other Pharmacopœias. It contains opium along with a number of other ingredients, chiefly aromatics, such as angelica root, serpentaria, valerian, squill, cinnamon, cardamoms, myrrh, flowers of sulphur, &c. One ounce contains about 5 grains of opium.—TRANSL.

jaundice which depend upon the impaction of concretions, and upon the spasmodic contraction around these masses of the muscular tissue of the bile ducts; their importance in other forms of jaundice appears very problematical.

6. The *acids*, such as the citric and acetic acids, chlorine water (*Siebert*), and especially nitro-hydrochloric acid, or aqua-regia.

The last of these was first recommended by Scott,* in the form of foot- and general-baths, and also for internal use, in jaundice and other rather obscure derangements of the functions of the liver, and, subsequently, it has been favourably spoken of by Annesley, Copland, and others. More recently, its effects in obstinate catarrhal jaundice have been extolled by Henoch, and justly so. The results which follow its employment in such cases, must be partly owing to the favourable action which it has over the relaxed mucous membrane of the stomach and duodenum, but principally to the influence which the acid ingesta in their passage from the stomach into the duodenum exercises over the excretion of bile. Claude Bernard has made the observation, which may easily be confirmed, that upon applying a glass rod, dipped in diluted acid, to the mouth of the ductus choledochus, a few drops of bile are squirted out, which is never the case when a weak alkaline solution is applied to the same spot.

The general effects of nitro-hydrochloric acid upon the metamorphosis of tissue, and the composition of the blood, have not yet been sufficiently cleared up; and hence, if we except its constricting effect upon the bile ducts, its value in the treatment of chronic affections of the liver, can only be purely empirical; the materials as yet in our possession are not sufficient for coming to a decision upon the matter.

7. The *Alkalies*, soda, potash, and carbonate of ammonia, have been particularly recommended in those forms of jaundice which depend upon impermeability of the bile ducts from thickening of the secretion, or the formation of concretions. It is hoped by their means to liquefy the bile and dissolve the concretions. How far this result may be expected from the alkaline carbonates remains to be seen. They are prescribed either by themselves, or in combination with the extract of rhubarb, aloes, &c.

8. The *Mineral Waters* of Karlsbad, Marienbad, Kissingen, Homburg, Vichy, Ems, &c.,† independently of the effects of the water

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* Medico. Chirurgical Transactions, Vol. VIII.

† The Springs at Karlsbad, in Bohemia, are thermal, their temperature

itself,—which, when taken in large quantity, finds its way through the walls of the portal vein and gives rise to an abundant secretion of thin bile,—are chiefly indebted for their action to the soda and neutral salts which they contain. They are with difficulty replaced by any other remedies, in cases where the jaundice owes its origin to chronic congestions of the liver, with obstinate catarrh of the bile ducts and mucous membrane of the stomach and duodenum, to gall-stones, &c. Their selection must always be determined by the nature of the fundamental affection of the liver, and by the constitution of the individual patient. The constant use of these waters is prejudicial in the case of new growths, such as cancer, or in more profound degenerations of the organ, such as cirrhosis, &c. They are

varying from 122° to 197° Fahr. They contain free carbonic acid, sulphate and carbonate of soda, chloride of sodium, the carbonates of lime and magnesia, with traces of iron, manganese, strontia, phosphate of lime, iodine, bromine, &c. They are much resorted to for biliary and calculous affections.

Marienbad is also in Bohemia, and only six leagues from Karlsbad. There are two principal springs at Marienbad, the Kreutzbrunn and the Ferdinandsbrunn. The former is the one most resorted to. It contains a large quantity of free carbonic acid, and, in the imperial pint, 75.442 grains of solid matter, which consists chiefly of sulphate of soda, with a little chloride of sodium and carbonate of soda, and a small quantity of carbonate of iron.

At Kissengen, in Bavaria, there are three springs, the most important of which is the Ragocszy. This contains a large quantity of free carbonic acid, and 95.76 grains of solid matter in the imperial pint. These solids consist, for the most part, of chloride of sodium (70.768 grains), with chloride of magnesium (7.543 gr.), carbonate of lime (4.034 gr.), carbonate of iron (0.707 gr.), bromide of magnesium (0.789 gr.), &c. The waters are laxative, tonic, and alterative.

Homburg, in Hesse, is about three leagues from Frankfort. There are four springs here, the most frequented of which is called the Elizabeth, which contains a very large quantity of chloride of sodium, and is strongly charged with carbonic acid.

The waters of Vichy, in central France, are perhaps the most celebrated in the world. They owe their virtues almost exclusively to bicarbonate of soda, which they contain in large quantity, along with much free carbonic acid. There are eight springs at Vichy, three of which are thermal, one of them, the Puits Carré, having a temperature of 113° Fahr.

The waters of Ems, in Nassau, are very similar to those of Vichy, containing a large quantity of bicarbonate of soda, with free carbonic acid. One of the springs, the Kesselbrunn, has a temperature of 114.8° Fahr.—TRANSL.

not, therefore, to be resorted to when there is any uncertainty in the diagnosis, or when the indication to be fulfilled in each individual case is not perfectly clear.

9. *The Varieties and Special Etiology of Jaundice.*

There have at all times been distinguished numerous varieties and forms of jaundice, which sometimes have been made to proceed from the causes of this condition being nearer or more remote, but at other times have been attributed to the period of life and to other circumstances of the patients. It would be a fruitless undertaking to examine more closely the various classifications which have arisen in this way, because in none of them can any fixed principle be laid hold of, and hence many of them have been gratuitous assumptions, or merely adapted to meet a practical necessity.

In all of these classifications, jaundice is considered as divisible into two great etiological groups. In the one, there may be detected structural changes of the liver, which furnish a sufficient explanation for the origin of the jaundice, which is then only a symptom of a disease of the liver. In the second group, there can be found no anatomical lesion to account for the development of jaundice through any impediment to the flow of bile; the ducts are here intact, and there is no obstruction to the egress of the secretion. Our views as to the origin of these forms of jaundice, are by no means clear; abnormal distribution of the hepatic secretion from impediments to the circulation of blood, or defective metamorphosis of bile in the blood, furnishes us with a ready explanation, which is certainly supported by facts, but by no means firmly established. Of the forms of jaundice belonging to this class, only a portion are intimately related to the proper functions of the liver; there are others which, on the contrary, proceed from a derangement in the consumption of bile in the blood, and are dependent upon influences which modify and interfere with the metamorphosis of matter, upon infection of the blood, upon derangements of respiration and of the heart's action, and, indirectly, through the last two, upon disorders of the nervous system.

Whether the etiological causes included in this category exhaust the various modes in which jaundice may originate, cannot at present be determined; it is difficult to find among them a definite place for some cases.

The complicated relations of jaundice to processes of a local and general nature just alluded to, offer a general classification of the different forms of the affection, as regards the peculiarities arising from the nature of their primary causes, which will smooth many difficulties in diagnosis in the subsequent chapters on diseases of the liver.

I. *Jaundice resulting from stoppage of bile, owing to diseases of the liver and bile ducts.*

The obstructions which impede or arrest the excretion of bile from the liver, are of a manifold nature, and are situated sometimes in the large ducts lying external to the liver, the ductus hepaticus and ductus choledochus; sometimes, on the contrary, they are situated in the smaller ducts within the gland; and lastly, at other times, they lie at the commencement of the bile ducts, at the circumference of the lobules. In all cases the passage of bile is obstructed, and jaundice results; but the intensity of the jaundice, as well as the other effects of the obstruction, may vary greatly.

A. *Jaundice in consequence of constriction of the ductus choledochus and hepaticus.*

The evacuation of the larger bile ducts is most frequently impeded by catarrh of their lining mucous membrane; *icterus catarrhalis* is the most common form of jaundice. It is ushered in with symptoms of catarrh of the stomach and bowels, which last for some days, and sometimes even for a longer period, before the yellow colour of the conjunctiva and of the skin can be distinguished. At the same time, in most cases, the hepatic region becomes painful upon pressure, the dimensions of the organ are increased, and the urine assumes a brownish colour, like beer, whilst the fæces become pale, and sometimes even every trace of the admixture of bile disappears from them. Slowness of the pulse and itchiness of the skin are frequent, but not constant phenomena. The appetite, in most cases, soon returns; after from eight to fourteen days, the stools again assume by degrees a dark colour, and after three or four weeks every trace of the disease has usually disappeared.

Occasionally, catarrh of the bile ducts is protracted, and gives rise to enlargements of the ducts and more intimate structural lesions of the hepatic tissue. (See Chapter on *Diseases of the Bile Ducts.*)

A similar impediment to the excretion of bile is observed to arise from compression of the bile ducts on the under surface of the liver, by the accumulation of fæcal matters in the large intestine, and by the enlargement of the uterus resulting from pregnancy. The latter, however, is very unusual, because, in addition to the enlargement of the uterus, it is necessary that it should take a certain direction in order to produce this accident. The possibility of such a compression has indeed been doubted, but unjustly so. I have repeatedly observed cases of jaundice attributable to considerable accumulations of fæcal matter in the colon, which could be circumscribed by means of palpation and percussion, and where purgatives effected such a speedy removal of the jaundice, that any other explanation of its origin, such for instance, as the existence of catarrh of the bile ducts, appeared inadmissible.

In like manner, jaundice may be produced by the lymphatic glands in the fissure of the liver, when these become enlarged from lardaceous (*speckig*), tubercular, or cancerous infiltrations. In this way, jaundice is not so very rarely an accompaniment of lardaceous liver, and it is a mistake, to make the absence of this symptom a diagnostic criterion of lardaceous degeneration of the liver, as has recently been done.

Constriction, or complete occlusion of the bile ducts, often results from an abnormal condition of their own contents, especially from concretions, less frequently from inspissated bile, and still less frequently from foreign bodies passing into the duct from the bowel.

That form which is produced by concretions, may almost always be recognised by the colic which characterises gall-stones, and, as a general rule, it terminates with the passage of one or several stones from the bowels. The jaundice attains a greater or less degree of intensity, according to the length of time that the gall-stone is impacted, and the completeness of the occlusion of the canal; the colour of the skin and conjunctiva is often slight, and after lasting a short time disappears, returning repeatedly after fresh attacks of colic. In exceptional cases, the stone remains impacted for a long period, and then there arise all those symptoms which are wont to accompany obliteration of the bile duct.

It has been repeatedly asserted, that inspissated bile can give rise to jaundice, although many have doubted the possibility of the affection originating in this way. Cases of this nature are certainly rare, and correspond in frequency to those cases in which, upon *post-mortem* examination, the bile is found to be thick and granular, and requires strong pressure to be applied to the gall-bladder, in order to squeeze it through the ductus choledochus into the bowel. That certain cases of this nature are met with, I believe I must conclude from one observation, in which, on the abatement of the jaundice, there were found in the clay-coloured stools brownish-black flakes, at first sparingly, but afterwards in increasing quantity, until the motions regained their normal colour. The gall-bladder which had previously been distended and painful, became collapsed, and all attempts to detect any concretions were unsuccessful.

In very rare instances foreign bodies of another nature are present in the bile ducts, or in the *diverticulum Vateri*,[*] and give rise to jaundice. To these belong the round worms, which creep from the bowel into the hepatic ducts;[†] and likewise the observation of Saunders,[‡] who found a currant-stone in the mouth of the ductus choledochus, where it opened into the bowel.

It is still questionable, whether clots of blood and solid fibrinous exudations, which, in exceptional cases, are present in the bile ducts, can produce such a persistent closure of these ducts, as to cause jaundice; at all events, I know of no examples of this nature.

B. *Jaundice in consequence of closure of the Ductus choledochus and D. hepaticus.*

This is sometimes the result of adhesions of the walls of the ducts, which may arise from exudative processes, but which in most cases are produced by the cicatrisation of ulcers of the mucous membrane; at other times, on the contrary, the occlusion is owing to the firm impaction of foreign bodies, or to the filling up of the canal by carcinomatous growths from its lining membrane; most frequently it is caused by pressure from without. This pressure may be exerted by newly-formed cords of areolar tissue, the products of inflammation of the hepato-duodenal ligament; or by cancerous tumours of the pylorus, of the duodenum, or of the head of the pancreas, or by tumours in the liver, growing outwards from its surface.[§]

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[*] The diverticulum formed by the junction of the ductus com. choledochus and the pancreatic duct.—TRANSL.

[†] Lieutaud (*Histor. anatom. med.*, I., p. 211), Roederer and Wagler, Cruveilhier (*Diction. de Méd. et Chir. prat.; Entoz.*), Lænnec (*ibid*), and Guersant (*Dict. de Méd.; Vers Intest.*), record observations of round worms in the bile ducts; still, it does not appear that jaundice was invariably present in these cases.

[‡] *Op. cit.*

[§] Job van Meckren records a case in which the bile ducts were closed

The complete impermeability of the bile ducts, which is produced in this way, and which very generally is of a persistent character, gives rise to jaundice of the most intense form. The jaundiced tint of the tissues and of the excretions here attains a degree which, under other circumstances, is by no means common. In consequence of the distention of the bile ducts, the volume of the liver is considerably increased, so that the lower border of the organ projects beyond the margin of the ribs, and may easily be felt on palpation. When the obstruction has its seat below the attachment of the cystic duct, the gall-bladder is also distended to a greater or less degree. In the cases which have come under my own observation, the gall-bladder has not contained above from eight to sixteen ounces of bile, but the older physicians mention cases, in which the quantity was much more considerable: thus De Jonge* found seven, and Van Swieten, eight pints of thick bile, accumulated in the gall-bladder. Upon careful examination, the smooth pear-shaped tumour of the bladder projecting beyond the margin of the liver is in general easily felt; in a few cases, it may be seen as a prominent tumour.†

The increase in the volume of the liver and of the gall-bladder usually goes on for some months; it then comes to a stand-still, and there ensues a more and more observable diminution. This is a proof that the secreting function of the gland is impaired. At the same time, unequivocal symptoms of deranged nutrition, and of obstructed portal circulation manifest themselves, the patients become flabby and thin, the digestion is embarrassed, water collects in the abdominal cavity, and sometimes hæmorrhages from the stomach and bowels occur. A fatal termination usually ensues, from the gradually increasing exhaustion, or from general dropsy, or from consecutive exudation processes; less frequently it takes place suddenly by peritonitis consequent upon perforation, and extravasation of

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by the pressure of an intussusception. Stokes (*Diseases of the Heart*, p. 638) mentions another, in which it was obstructed by an aneurism of the hepatic artery.

* Philosoph. Transac., Vol. XXVII.

† I am at present attending a lady whose gall-bladder reaches downwards to 1½ inch below the crest of the ilium, and elevates the abdominal wall in the form of a pear-shaped tumour. Owing to the great tension and pain which it occasioned, it became necessary to puncture it; and, from the presence of adhesions, there was no danger in effecting this. About ten ounces of bile flowed out.

bile; or death may take place under symptoms of suppurative fever, induced by suppuration of the liver. In some cases, the complete destruction of the hepatic functions leads to cholæmic intoxication.

c. *Constriction, or Obstruction of the Bile Ducts within the Liver.*

It is from the presence of one or other of these conditions that jaundice becomes a frequent symptom of hepatic diseases. All morbid alterations of the liver, rendering the larger branches of the excretory ducts impermeable, such as cancer, echinococci, inflammatory deposits, &c., induce jaundice, which is always greater in intensity, the more extensive the volume of the liver, and the greater the number of ducts, which are implicated. Hence tumours or inflammations on the concave surface of the organ are usually accompanied by jaundice, which is generally wanting when these same lesions have their seat on the convex portion, or in the posterior part of the right lobe. It is seldom that the bile is in this way completely excluded from the intestinal tube, in cases where the new growth, or the inflammatory deposit, does not implicate the hepatic duct; the fæces almost invariably exhibit an admixture of bile. It is worthy of notice, that the jaundice which arises in this way, although there is no corresponding variation in its generating cause, varies in intensity at different times, according to the variations produced upon the bulk of the tumour, as well as upon the secreting functions of the liver and the compensating functions of the kidneys, by the changes in the quantity of blood supplied to these parts. This circumstance must not mislead our diagnosis, which, indeed, is seldom difficult, inasmuch as, in most cases, the situation of the tumour can be detected by palpation.

The jaundice which is produced by the compression of the bile ducts near their origin is extremely slight. This form is now and then observed as an accompaniment of cirrhosis. Under such circumstances, the results of an obstruction to the excretion are usually limited to a more or less intense jaundice of the hepatic parenchyma, the colour of the skin, conjunctiva, and urine, remaining unchanged. In many cases, however, there may be observed a light-yellow tinge of the conjunctiva, and a darker colour around the eyes, while brownish spots may be seen at the angles of the mouth, upon the forehead, on the temples, and on other parts of the body; at a later period, the pale skin assumes throughout a yellowish colour.

The stools are sometimes clay-coloured, sometimes brown, and there may be often seen in the evacuations dark normally-coloured masses, mixed up with others which are pale and free from bile. Sometimes the urine contains pigment; at other times it does not.

The expansion of the hepatic cells in fatty liver operates in obstructing the excretion of bile in a similar manner to that in which the areolar tissue acts in cirrhosis; but it is rare for general jaundice to result in the former case. A similar result arises from considerable congestions of the liver, as happens when there is obstruction to the circulation in consequence of disease of the heart, lateral distortion of the chest, &c., the origins of the bile ducts being then compressed by the enlarged capillaries. The jaundice, in most cases of this nature, is limited to a light-yellow tinge of the conjunctiva and skin, and becomes more distinct after violent attacks of dyspnœa. The yellow colour is easily overlooked when there is a florid or livid complexion. (*Icterus plethoricus* of the ancients.)*

The following observations may be given here as illustrations of the points just discussed:—

OBSERVATION No. V.

Dyspepsia.—Symptoms of Chronic Simple Ulcer of the Stomach.—Jaundice.—Distention of the Gall-bladder.—Pleurisy of the right side.—Dropsy.—Petechiæ.—Death.

Cancer of the Duodenum and Dilatation of the Bile Ducts.—Simple Ulcer of the Stomach.—Exudation in the Right Pleural Cavity.

Marianne Dombrowsky, a female servant, aged 63, was admitted on December 13th, 1853. The patient had been complaining for a year of pains in the epigastrium, which occurred after eating, and were accompanied by heartburn, nausea, and sometimes even vomiting; subsequently, the appetite had completely failed, whilst the pains had extended over the hepatic region. Four weeks before, jaundice of the skin had gradually become developed.

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* Stokes (*Diseases of the Heart*, p. 206) has observed repeated attacks of jaundice and hemiplegia to result from incompetence of the mitral valves, these symptoms on each occasion disappearing at the expiration of one day, under the use of stimulants. Not unfrequently, the jaundice, in cases of disease of the heart, is the consequence of catarrh of the duodenum, and then it is of longer duration.

On admission, the patient was emaciated, her skin was dry and flabby, and tinged yellow to a considerable degree. The thoracic organs exhibited nothing abnormal, with the exception of well-marked emphysema of the lungs, especially of the right one. The abdomen was soft, and some portions of the bowel were distended and tympanitic. The liver was deeply situated; its upper border close to the sternum was on a level with the seventh rib; the outline of the organ was somewhat increased; its dulness on percussion, in the sternal line amounted to 12 centimètres; in the mammary, to 15; and in the axillary, to 13. (4¾, 6, 5⅛ Eng. inches.) Close to the umbilicus, on the right side, and two centimètres (¾ inch) from the surface, there might be felt a rounded, painful tumour, which sank downwards upon a deep inspiration, and could be traced upwards as high as the sharp margin of the liver. The upper and under surfaces of the liver, so far as could be made out by palpation, felt smooth and even. No hard tumour could be detected either upon the liver or in the direction of the ductus choledochus as far as the duodeno-pyloric region, even after repeated and careful examinations, with the abdominal parietes relaxed, and with the patient in different positions.

The spleen was somewhat enlarged. The stools were clay-coloured and smelt badly. The urine was coloured brown like porter, and when spread out in thin layers, was saffron-yellow.

The pulse was 60 and weak. There was no itchiness of the skin.

The patient was ordered infusion of rhubarb with the watery extract of nux vomica, and ethereal tincture of valerian. The appetite improved, the pains during digestion, as also the flatulence, were diminished, the motions became regular, but the stools still remained free from bile-pigment. The distended gall-bladder increased in size, but continued smooth, painful and moveable.

Three weeks after admission, dulness on percussion and absence of the respiratory murmur were detected at the base of the right lung, and extending upwards as high as the fourth rib; at the same time, the bronchial tubes were observed to become loaded with mucus, the respiration became more frequent, and the patient began to complain of dyspnœa, and expectorated with difficulty rounded masses of tenacious mucus. The pulse rose from 80 to 85. The urinary secretion was diminished.

Decoction of senega and liquor ammoniaci anis.* were prescribed.

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* The *Liquor Ammoniaci anisatus* of the Prussian Pharmacopœia contains

A turpentine liniment was ordered to be rubbed over the thorax and abdomen; and in the evening, 2 grains of the watery extract of aloes were administered, to open the bowels.

The patient became rapidly collapsed; œdema appeared in the feet, and in a few days extended to the pelvis. The abdomen became fluctuating and tender upon pressure. The pulse was small, and 110 in the minute.

On the 26th of January she lost her consciousness, the stools were passed involuntarily, and there were hiccup, dilated pupils, and stertorous respiration. Numerous petechiæ, varying in size from that of a linseed, to that of a groschen piece,* made their appearance upon the skin of the trunk and extremities.

Death supervened on the 27th, at 9 A.M.

Autopsy, 26 *hours after death.*

The skull cap and dura mater were coloured yellow, but otherwise normal; there was a small quantity of coagulated blood in the longitudinal sinus. The arachnoid, together with the longitudinal sinus, was considerably thickened. The pia mater was of a blood-red colour. There was a considerable quantity of clear yellow serum at the base of the skull, and in the lateral ventricles. The brain substance contained but a small quantity of blood, it was of normal consistence, and no yellow tinge could be detected in it.

The mucous membrane of the pharynx and bronchi was slightly injected. The epiglottis was of a saffron-yellow colour.

The left side of the thorax contained about a pound of clear yellow serum; and in the right side there were about four pounds of fluid mixed with fibrinous flakes. The upper part of the left lung was dry and emphysematous, the lower part was compressed; the right lung was very emphysematous, its lower lobe was almost completely compressed.

There were three ounces of clear yellow fluid in the pericardial sac; the epicardium exhibited a jaundiced tint; the cavities of the heart contained some coagulated blood; its valves and muscular tissue were normal.

Upon opening the abdomen, the stomach was found to be dis-

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rectified spirit (32 parts), liquor ammoniæ (8 parts), and anise oil (1 part), —TRANSL.

* A silver groschen is somewhat smaller than a sixpence.—TRANSL.

tended with gas, so that the liver was partially covered by it, and pushed towards the right side; close to the pylorus might be seen the distended gall-bladder. The transverse colon was firmly adherent to the liver, and the posterior part of the surface of the gall-bladder. It here formed an angular coil in such a way, that the curvature lying underneath the gall-bladder first took a turn backwards and to the right, and afterwards passed towards the left hypochondrium, along the great curvature of the stomach; there was also an angularly bent coil of the sigmoid flexure, firmly glued together by recent areolar tissue. In the abdominal cavity there was found a large quantity of serum mixed with fibrinous flakes.* The gall-bladder

FIG. 21.

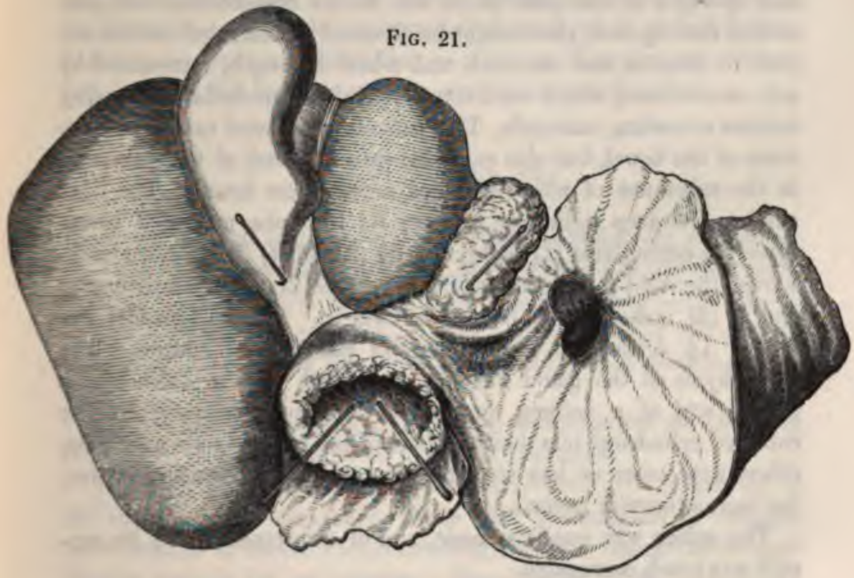

FIG. 21. An ulcerated cancerous deposit in the duodenum passing into the head of the pancreas. Probes passed into the pancreatic and common bile ducts are seen to emerge in the centre of the cancerous mass in the duodenum. Upon the mucous membrane of the stomach there is represented a simple ulcer with an adherent clot of blood.

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* This exudation had a feeble alkaline reaction, and a greenish colour. Upon the addition of acetic acid to the filtered fluid, there was deposited a precipitate which was insoluble in an excess of the acid. Bile-pigment

was loosened from its adhesions and the ductus choledochus laid bare; this was distended to the breadth of an inch, as far as its insertion into the duodenum. The stomach contained a brownish-black, greasy fluid; about an inch and a-half below the cardia there lay upon its posterior wall an oval simple ulcer, almost the size of an eight-groschen (halfpennypiece), and attached to its surface was a blackish tumour, the size of a pigeon's egg, consisting of a firm coagulum of blood. Excepting this, the mucous membrane of the stomach, as well as of the pylorus, showed nothing abnormal. The mucous membrane in the upper part of the duodenum was thickened and spongy; at the part where the ductus choledochus and pancreatic duct opened, there might be observed an ulcerated surface one inch in breadth, and one inch and a-half in length, surrounded by soft excrescences, which were connected with a medullary fungating tumour extending outwards. This tumour penetrated not merely the coats of the bowel, but also extended into the head of the pancreas, in the substance of which softened masses were found. The liver exhibited a deep tight-lace furrow; its left lobe measured transversely 3 centimètres (1⅕ inch), and 5¼ (2 inches) from before backwards; the right lobe measured 6 centimètres (2⅓ inches) transversely, and 8 (3⅐ inches) from before backwards. The gall-bladder extended 1½ centimètre (⅔ inch) beyond the anterior margin. The parenchyma of the gland was firm and hard, of a greenish-brown colour, and of a nutmeg appearance, and was perforated by the enlarged cylindrical bile ducts. Some of the hepatic cells were pale, others were more or less strongly saturated with colouring-matter, but were otherwise normal.

The spleen was small, anæmic, and of firm consistence; its capsule was much corrugated.

The pancreas was normal.

The intestinal canal contained greyish clay-coloured fæces, its mucous membrane was unaltered.

The kidneys were of normal size; their parenchyma was of a jaundiced tint, but free from deposits of solid pigment. Their more intimate structure exhibited nothing abnormal, with the exception of coloured epithelium. The mucous membrane of the pelves of the kidneys, of the ureters, of the bladder, and of the vagina was of

was found to be present on testing with nitric acid; the biliary acids and sugar could not be extracted by diluted spirit of wine, from the dried residuum.

a saffron-yellow colour. In the situation of the uterus were found several aggregated rounded tumours, which had replaced the substance of the organ; one of these was of stony hardness and of the size of an apple. These tumours also were coloured yellow.

Kölliker* has recently ascertained, that calcareous matter is deposited in the intestinal arteries, and simple ulcers formed in the stomach of animals, after the application of a ligature to the ductus choledochus; and he has hinted at the possibility of a dependence of these conditions on the retention of bile. In the preceding case, the gastric ulcer had already existed before the commencement of the jaundice, at least the symptoms indicative of it preceded this; hence it cannot be concluded that, in this case, there was any connection between the jaundice and the ulcer as cause and effect.

Observation No. VI.

Cancerous Deposit in the head of the Pancreas and in the Duodenum.—Occlusion and widening of the Bile ducts and of the Pancreatic ducts.—Distention of the Bile ducts with mucus and bile.—Dysentery.—Diminished secretion of urine.—Infiltration of the kidneys with solid deposits of bile-pigment.—Death from Exhaustion.

Carl Bohle, a day-labourer, aged 55, was admitted on the 27th November, 1854, and died on the 10th December.

The patient, who had enjoyed uninterrupted good health up to six months before admission, began then to complain of frequent short attacks of severe pain, extending from the region of the gall-bladder towards the epigastrium. Seven weeks before, without any other derangement of the general health, he gradually became jaundiced, and, for four weeks he had been treated as an out-patient, with rhubarb, aloes, &c.

On admission, the man was of robust habit of body, his skin was coloured a brownish-yellow, and, when spoken to, he gave slow and surly, but rational answers. The thoracic organs were normal; the pulse 64, and weak; the tongue coated greyish-brown; the appetite poor, and the abdomen was somewhat tympanitic and contained a considerable quantity of fluid. The liver was slightly enlarged; its dulness in the median line amounted to 8 centimètres; in the mammary line, to 15; and in the axillary, to 12. (3⅐, 6 and 4¾ inches). On palpation, its surface appeared smooth, without any tuberosity, and its margin was sharp. At the outer border of the right rectus abdominis muscle, at the same elevation as the umbilicus, a smooth, pear-shaped, moveable tumour could be felt, which could be traced up under the margin of the liver. No hard tumour could be detected in the region traversed by the ductus choledochus in its passage to the duodenum. Hence, bearing in mind the early attacks of colic, it was reasonable to attribute the cause of the dilatation of the bile ducts and of the jaundice to an occlusion of the ductus choledochus by means of a concretion. Still it was impossible to make a diagnosis with positive certainty, as to the cause of the obstruction of the bile duct.

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* Würzburger Verhandl., Bd. VI., S. 474.

The patient took calomel and opium up to the 2nd of December. The stools were now passed regularly; they lost their grey colour and became greenish. No gall-stones, however, could be found.

In order to avoid salivation, the mercury was discontinued, and the bowels were kept open by means of aloes; and compound tincture of bark with naphtha aceti * was prescribed, with the object of stimulating the sluggish digestion.

The skin gradually assumed a bronze colour, and the urine became brownish-black. Its quantity was small, and, although the patient complained of some thirst, gradually diminished more and more. The colour of the stools was somewhat brownish, not owing, however, to the admixture of bile-pigment, but, as shown by the microscope, to numerous epithelial cells containing pigment, derived from the mucous membrane and glands of the intestine.

From the 4th of December the patient became rapidly more emaciated, his strength sank, and his appetite completely failed; he had also frequent bloody stools, with tenesmus; pulse 70, and weak. The mental faculties became impaired, and he scarcely answered when spoken to. The urine, so far as its quantity could be ascertained, was very scanty, in consequence of the frequent dysenteric stools.

During the last night of the patient's life, he was very restless, made attempts to get out of bed, and became more exhausted.

He died on the 10th of December, at 6½ A.M.

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* Naphtha aceti is the æther aceticus of the Prussian Pharmacopœia, or acetic ether.—TRANSL.

Autopsy, 6 *hours after death.*

The skin of the dead body was brownish-yellow, and at some places bronze-coloured, as, for instance, on the inner surface of the thighs, whilst the countenance was paler. There was no œdema.

The skull-cap was 7 Paris lines thick, with but little diploe. The dura mater was yellow and thickened; beneath it a thin extravasation of blood was found, extending over both hemispheres: the brain substance was of normal colour and consistence.

The mucous membrane of the pharynx and œsophagus was of a jaundiced colour, but otherwise normal. The bronchi presented a similar appearance; both lungs were emphysematous at their apices and anterior margins, and posteriorly and inferiorly exhibited hypostatic congestion. The right cavities of the heart contained firmly coagulated masses of fibrin insinuated between the trabeculæ. The muscular tissue and valvular apparatus on both sides were normal; the endocardium was coloured dark-yellow.

Two pounds of reddish-brown fluid, exhibiting the reaction of bile-pigment, were found in the peritoneal cavity.

The relations of the liver closely corresponded to what had been ascertained by means of percussion and palpation on the patient's admission. Its volume was slightly increased. Its surface was smooth, and its margin sharp. The organ was removed along with the neighbouring parts, so as to ascertain the relations of the bile ducts *in situ*. These ducts were enormously enlarged; the ductus choledochus just before its entrance into the duodenum measured an inch and a-half in its transverse diameter; the cystic duct, the gall-bladder and the hepatic ducts were distended in a corresponding manner. The cystic duct measured 11 Paris lines; the hepatic, 22; and one duct in the right lobe, 17. They were full of a fluid presenting a whitish turbidity; but otherwise transparent. This was feebly alkaline, and, on the addition of nitric acid, presented no trace of the reaction of the colouring-matter of bile; neither could the bile acids be detected by means of Pettenkofer's test.* The usual epithelial lining of the bile ducts could nowhere be discovered upon microscopic examination; but in place of it, there were seen globular mucous corpuscles either isolated or adhering in great numbers in epithelial-like laminæ. Nitric acid when added to this fluid produced no precipitate, or one that was scarcely appreciable. On the addition of acetic acid, the fluid became thickish and resembled bile. The addition of water produced a slight turbidity. The

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* In Pettenkofer's test, advantage is taken of the circumstance, that various colours are developed when sulphuric acid and sugar are added to a solution containing any of the bile acids. The fluid to be tested is first evaporated almost to dryness, and the residue extracted with alcohol. The alcoholic solution is again evaporated, and the residue dissolved in a little

FIG. 22.

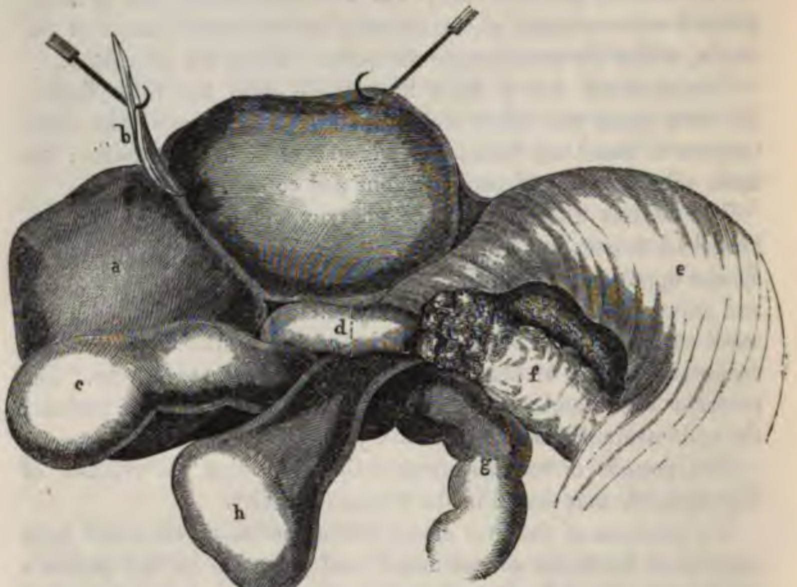

FIG. 22. Represents an enormous enlargement of the bile ducts, resulting from cancerous deposit in the head of the pancreas: *a*, the liver turned back; *b*, ligamentum teres; *c*, the greatly distended gall-bladder; *d*, the dilated ductus choledochus; *e*, the stomach; *f*, the pancreas; *g*, the duodenum; *h*, the right kidney.

water and placed in a white Berlin china dish. A few drops of a solution of sugar are now to be intimately mixed with the solution in the china dish, and concentrated sulphuric acid added to this mixture in drops. When biliary acids are present, the solution at first becomes a little turbid, then clears, and afterwards assumes a pale cherry-red, then a pink, then a crimson, and at last a saturated purple-violet hue. The intensity of the colours depends upon the quantity of the biliary acids present.—TRANSL.

fluid when dried at a temperature of 100° cent. (212° Fahr.) left behind 1.80 per cent. of solid residuum, of which 1.15 consisted of ash, and 0.65 of organic matter; the ash contained 0.08 of earths insoluble in water, and 0.07 of alkaline salts. The composition of the fluid may be represented in a tabular form, thus:—

Water	98.20
Solids	1.80
	100.
Ash	1.15
Organic matter	65
Total solids	1.80
The ash contained—	
Alkalies	0.07
Earths	0.08

The gall-bladder, in addition to a few very small mulberry concretions, contained a fluid precisely similar to that just described, with 1.72 per cent. of solid residuum.

The parenchyma of the liver was impregnated with a brown colour, scattered through which might be observed the congested ramifications of the hepatic veins. In fine sections of a portion of the organ, after this had been boiled and rendered transparent by a weak solution of caustic potash, numerous enlarged bile ducts with thickened walls could be distinguished, some of them rounded, and others ampulliform. (See Fig. 20, page 116.) In many parts of the liver, these ducts might be traced as far as the periphery of the lobules; but in others, this could not be done. No communication could be made out between the blunted and somewhat club-shaped extremities of the distended ducts, and the more delicate channels in the interior of the lobules, from which these ducts take their origin. This even could not be traced in those places where the sections had passed longitudinally through the distended ducts; in such a case, the sections of the tubes presented distinct sacculated bulgings, but each duct invariably terminated bluntly at the periphery of the lobule, without any finer offshoots.

The hepatic cells were for the most part normal in their characters; they appeared pale, contained but a small quantity of granular matter, and the nucleus was distinctly visible only in a few. Some of the cells had pigment deposited in them in the form of isolated brown molecules in the neighbourhood of the nucleus, or round the circumference of the cell; these molecules were here and there aggregated in clusters, and occasionally formed large greenish spherical masses like drops. Some of the cells were of a uniform brown or greenish colour. Along with these were observed little elongated bodies, shaped like sausages, of a brown or bluish green colour, which were for the most part swimming freely about, but occasionally were adherent to the side of a cell: less frequently these bodies were observed to be globular, or ramified in an arborescent manner, or sharply angular.

The large vessels of the liver, the portal vein, the hepatic veins, and the hepatic artery, did not exhibit anything abnormal. The spleen was small and weighed 0.14 kilogram. (5 oz. avoird.); it was wrinkled, contained but little blood, and was of good consistence. The mucous membrane of the stomach was pale-yellow, but otherwise normal. The head of the pancreas was enlarged, and occupied by a mass of scirrhus. The cancer had completely obliterated the ductus choledochus, and extended into the duodenum in the form of a pendulous tumour the size of a walnut. The pancreatic duct was also occluded, and at the same time enlarged to the extent of 4 Paris lines in its transverse diameter. (Fig. 23.)

The mucous membrane of the small intestine was coloured bluish-green, owing to the epithelium of the villi and of the glandular epithelium of Lieberkuhn's follicles being loaded with black pigment. The mucous membrane of the rectum was tumid and covered with ecchymoses; this puffiness was still more remarkable at the second curve of the colon and in its transverse portion; no dysenteric ulcerations were visible after the removal of the bloody mucus.

The kidneys were of an olive-green colour. Upon their outer surface there were seen numerous brownish-black ramifications corresponding in their extent and course with the tortuous uriniferous tubes. These were much more distinctly visible on a cut surface, especially in the pyramids. Here many of the tubes resembled long black stripes, and along with these there were others of a brown, sap-green, or yellow colour. The microscope afforded a deeper insight into this interesting, but hitherto unsufficiently studied, alteration of the renal parenchyma.

The paler uriniferous tubes were of a uniform green or brown colour; their epithelial cells, which, in a few instances only, were found entire (and these, as a general rule, were the ones in which there was the least pigment), exhibited a deep brown colour; this was darkest in the nuclei. Some of the cells appeared tinged blood-red; others were rounded, and contained brown or green colouring-matter, deposited in concentric layers around the nucleus. Here and there might be seen epithelial cells in a state of fatty degeneration, some of them red, and others brown or black. In many places brown cell-nuclei, either isolated or adhering in groups, lay upon the wrinkled, greenish-yellow basement-membrane of the tubuli; at other places this membrane was covered by a mass of fine brown granules. Where the deposit of the constituents of bile was most intense, the little tubes were filled with a black, brittle material, which was only slowly and incompletely dissolved on the addition of caustic potash, the solution being of a brown colour. There might also be seen a few small cylindrical masses, consisting of an amorphous, dark-brown

FIG. 23.

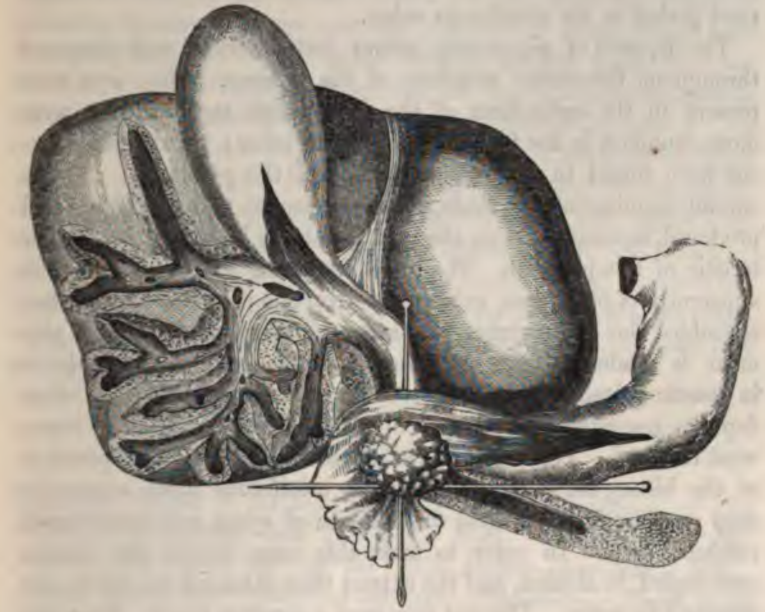

FIG. 23. Cancer of the head of the pancreas, penetrating, in the form of a pendulous tumour, into the interior of the duodenum, and producing occlusion, and consequent dilatation of the pancreatic and bile ducts.

substance, gradually becoming paler towards the periphery. Caustic potash acted rapidly upon these masses; their pigment disappeared; they became pale, transparent, and swollen up; and they then resembled old fibrinous clots which had been deposited for a lengthened period in the uriniferous tubes.

The deposits of pigmentary matter just described were scattered throughout the entire structure of the kidneys. They were even present in the epithelium of the Malpighian capsules, but were most abundant in the tortuous uriniferous tubes; the darkest deposits were found in the straight tubuli of the pyramids. A more careful examination was made as to their precise nature. Nitric acid produced, in most of them, the play of colours known to be characteristic of bile-pigment. The black crumbling masses, which were apparently of older date, exhibited no reaction; in this respect they resembled the cholepyrrhin of black gall-stones, in which the pigment is gradually more and more altered, becomes less soluble in caustic potash, and loses its reaction with nitric acid. The other deposits consisted of bile-pigment, partly recent, and partly somewhat changed. Pettenkofer's test appeared to indicate the presence of the biliary acids, inasmuch as the uriniferous tubes assumed a deep purple-red colour upon the addition of syrup and concentrated sulphuric acid. In order to make this more certain, the kidneys were boiled in alcohol, and the extract thus obtained was dried and dissolved in water. The test now gave a negative result. Even the alcoholic solution did not present the characteristic reaction. Hence it could not be assumed that the biliary acids were present in the kidneys.

The urinary bladder contained a little dark urine; its mucous membrane was jaundiced. The retro-peritoneal glands were yellow and infiltrated with cancerous matter. The firmly coagulated blood taken from the right side of the heart, was examined for the elements of bile; pigment was found, but not a trace of the biliary acids or of their immediate derivatives.

The urine, passed by the patient between the 2nd and the 7th of December, was repeatedly examined. It was of a dark, brownish-green colour, acid, and deposited, on standing, a light flocculent sediment, mixed with brownish-black, angular granules; albumen and sugar were repeatedly sought for, but never detected. It exhibited distinctly the play of colours when treated with nitric acid; but none of the other ingredients of bile could be found in it, except a few plates of cholesterine which were observed in the alcoholic extract of the secretion.

This case is interesting in several respects; first, because the deposit of bile-pigment in the kidneys had attained a rare degree of intensity, inasmuch as the secreting functions of these glands were essentially impaired; and further, because the bile ducts were not filled with bile, as is usually the case, but with a colourless mucous fluid. A similar case has already been recorded by De Graaf.* All communication between the bile ducts and the hepatic parenchyma was here cut off, so that no new secretion could pass into the ducts after their original contents had become absorbed, and replaced by the mucous secretion of their lining membrane. Thus, whilst almost all the solids and fluids of the body were tinged yellow, the contents of the hepatic ducts were colourless;—a proof that the mucous membrane of these ducts plays no part in the separation of bile from the blood.

OBSERVATION No. VII.

Cancerous Deposit in the head of the Pancreas.—Occlusion of the Ductus Choledochus and Pancreatic Duct.—Enlargement of the Pancreatic Duct, and of the Bile Ducts.—Jaundice.—Intestinal hæmorrhage.—Diabetes mellitus.—Dysentery.—Death from exhaustion.

Wilhelm Vogel, mason, aged 50, was admitted into the Clinique, on the 13th February, 1854, and died on the 9th of April.

The patient stated that he had suffered, for the period of a year, from pains in the upper part of the abdomen, which, however, had been transient and had attracted little attention, inasmuch as they produced no remarkable impairment of the general health. Since the beginning of December, or for nearly three months, the skin had been gradually assuming a jaundiced tint, and at the same time the pains in the hepatic region, although still only transient, had become more violent and extended towards the right shoulder. Moreover, tar-like masses were repeatedly observed in the stools, which could not be accounted for by anything in the patient's food.

On admission, the patient's skin was of a yellow-brown colour; his abdomen was soft and somewhat distended, but not tender upon pressure. The liver was more deeply situated than usual, and it was somewhat increased in volume. The dulness on percussion amounted in the sternal line to 13 centimètres, in the mammary to 12, and in the axillary line to 11 ($5\frac{1}{8}$, $4\frac{3}{4}$ and $4\frac{2}{3}$ inches). The sharp margin of the organ could be felt about two inches below the false ribs; on the left side, this margin was thin and could be pressed inwards; tracing it along towards the right, we arrived, at a distance of two and a-half inches to the right of the linea alba, at a pear-shaped, smooth, elastic swelling, the position of which was altered by the respiratory movements, and which was tender upon pressure. The swelling reached seven centimètres ($2\frac{3}{4}$ inches) beyond the margin of the liver. When the hand was firmly pressed immediately to the left of, and a little above, this swelling, it came upon a hard, nodulated, immoveable tumour, the form of which, however, could not accurately be made out. A tarry motion, evidently due to the presence of blood, which had just been passed, indicated a hæmorrhage high up in the intestinal canal. The appetite was unimpaired; there was no vomiting; and the pulse was 60, and weak.

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* Tract. de succo pancreatico, cap. 8.

The heart was normal. Harsh vesicular breathing and bubbling *râles* (*Rasselgeräusche*) could be heard over the apices of both lungs, and there was a limited dulness on percussion.

The urine was brownish-green, free from albumen, and of average quantity.

The diagnosis necessarily arrived at was, that there was occlusion of the ductus choledochus from cancerous deposit in the head of the pancreas, which, probably, also involved the pylorus. The opinion that the new growth was situated in the pancreas, was favoured by its situation, its complete immobility, and by the circumstance of its form being more globular than is wont to be the case in simple cancer of the pylorus. The existence of the latter lesion was also contra-indicated by the absence of vomiting, and by the fact of the ductus choledochus being involved, which is seldom the case in tumours of the pylorus. The tarry character of the stools, which was from time to time observed, appeared to indicate that either the walls of the stomach, in the neighbourhood of the pylorus, or those of the duodenum, were implicated. We did not, in coming to this conclusion, conceal from ourselves the fact that the hæmorrhage from cancer is usually not so copious as to blacken the stools, nor the circumstance that it is wont to be more persistent than it was in this case, where there were long intervals between the different attacks.

Under the use of rhubarb, aloes, and the ammonio-chloride of iron, the patient's condition remained for six weeks unchanged in its principal features, except that the emaciation increased, notwithstanding a good appetite and abundance of food. The hæmorrhage from the bowel did not return; the stools were clay-coloured without any admixture of bile.

From the 15th of March, it was observed that the jaundiced colour of the skin became paler, without any bile appearing in the stools. This appeared to depend upon an increased secretion of urine, which, by degrees, became doubled in quantity and lighter in colour; on closer examination, an abundance of sugar was detected in this fluid. The specific gravity varied from 1009 to 1018. Repeated careful examinations of the urine were now made, and with the following results:—*

Quantity of Urine in 24 hours. English Fluid Ounces.†		Specific Gravity determined by means of weighing, and the Gravimeter.		Per centage of Sugar.	Remarks.
Date.	Fluid Ounces.	Heavier Urine of Night.	Lighter Urine of Day.		
March 26 to 27	169.	1018.5	1012.		The morning urine was the heaviest. It contained most bile-pigment, and much mucus, and gave the reaction of sugar.
„ 27 to 28	172.5	1017.	1011.5	The lighter urine (1011.5) contained 1.28 p.c.	
„ 28 to 29	176.	1013.	1010.5		
„ 29 to 30	165.5	1014.5-1015	1010.5	2.88 p.c. (heavier, 1015) 1.08 p.c. (lighter, 1010.5)	
„ 30 to 31	158.5	1014.5	1009.	2.65 (heavier) 1.00 (lighter)	
„ 31 to Ap. 1	134.	1014.	1014.	2.45	
April 1 to 2	120.	1019.	1010.	3.8 (heavier)	
„ 2 to 3	98.55	1018.	1009.5	0.822 (lighter) Polariscope.	

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* To determine the quantity of sugar, the urine was diluted with nineteen volumes of water, and agitated with a boiling, always recently-prepared, solution of sulphate of copper, tartaric acid, and potash, containing a fixed quantity of copper, until a few drops of the filtered fluid yielded no red precipitate with hydrochloric acid and cyanide of potassium, and did not any longer precipitate the copper solution. The average result of three experiments was taken.

† These have been calculated from cubic centimètres in the original. The specific gravity is remarkably low for diabetic urine; but Frerichs informs me that he has occasionally observed such cases.—TRANSL.

The patient used opium in large doses* to counteract the tormenting loss of sleep and the diuresis; and to keep the bowels open the watery extract of aloes, with steel, was prescribed; attention was paid to the diet, which consisted principally of animal food.

This change in his diet, Vogel, who in other respects was a quiet and sensible man, found to be insupportable, and, consequently, on the 2nd day of April he demanded his discharge. The distention of the gall-bladder, the hard tumour close to the left of this organ, and the size of the liver remained the same as on admission.

So soon as the 6th of April, the patient returned in a much altered condition. His features were pale and collapsed; frequent evacuations of the bowels, consisting of mucus, blood, and fibrinous flakes, and accompanied by troublesome tenesmus, had quite exhausted him; along with this, the pulse was 64 and weak, the extremities were cool, there was complete loss of appetite, an inclination to somnolence, and slow answers. From the 7th to the 8th of April, 2,500 cub. cent. (88 fluid ounces) of urine were passed, of which the heaviest had a specific gravity of 1008, and the lightest 1005. It had an acid reaction, and contained bile-pigment, but not a trace of sugar. The quantity of urine passed from the 8th to the 9th, amounted to 2000 cubic centimètres (70 fluid ounces); had a specific gravity of from 1008 to 1006, and likewise contained no sugar.

An enema containing nitrate of silver and opium, and, internally, compound tincture of bark with ether, wine, soups, and other analeptics, failed in arresting the dysentery and exhaustion. Death ensued at one in the morning of April 9th.

Autopsy, 10 *hours after death.*

The body was jaundice-coloured to a considerable degree; there was no œdema. The dura mater was yellow, the pia mater was moderately congested, as was also the brain substance, which was somewhat softened, especially in the fornix, the corpora quadragemina, the pons Varolii, and the roof of the fourth ventricle. In the substance of the pons Varolii numerous little reddish-brown masses of pigment, the remains of capillary apoplexies, were found.

The mucous membrane of the mouth and pharynx, and also of the œsophagus was yellow; that of the larynx and air-passages was faintly injected. The apex of the left lung contained tubercular deposits of old date, which were surrounded by thickened tissue containing dark pigment, and, in the immediate neighbourhood, were portions of the organ in an emphysematous condition; the lower lobe was in a state of hypostatic congestion. The right lung, likewise, contained some tubercular masses, the size of a bean.

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* Three grains of opium were insufficient to induce sleep.

In the pericardium there were found five ounces of fluid tinged with bile, and highly albuminous, from which, soon after its removal, a quantity of fibrinous matter separated in the form of a cake. It contained abundance of bile-pigment; but sugar, and the biliary acids could not be detected, either directly or in the alcoholic extract of the residuum which remained after drying. There were firm, fibrinous coagula in both sides of the heart; the muscular tissue and the valvular apparatus were normal.

The peritoneal sac contained a pound and a-half of fluid, which was paler than that in the pericardium; it was turbid owing to the presence of pus; the reaction of the colouring-matter of bile was distinct, but more feeble than was the case with the pericardial effusion. Pettenkofer's test for the biliary acids gave a positive, and Trommer's test for sugar, a negative result.

The spleen was adherent to the flexure of the colon; its size was normal (4¾ Paris inches long; 3 inches broad, and ¾ of an inch thick); its capsule was thickened, and its parenchyma was soft and moderately congested.

The liver was situated farther down than usual. The anterior margin of the left lobe lay 3½ inches below the apex of the ensiform cartilage of the sternum, and that of the right lobe extended 2 inches beyond the cartilages of the eighth and ninth ribs, and the apex of the gall-bladder, fully 1 inch beyond this; the edge of the gall-bladder was 2½ inches distant from the median line.

The size of the liver was unaltered; its margins were sharp; and its surface smooth. The gall-bladder was enormously distended; it contained about eleven ounces of thick, brownish-black bile, in which there glistened many large and remarkably thick plates of cholesterine, but which contained no albumen. The bile ducts were all much dilated, so that at many places over the outer surface of the liver they could be felt to fluctuate. (See Fig. 19, page 115.) Their mucous lining had lost its cylindrical epithelium, and was covered with pavement epithelium, which had partly undergone fatty degeneration. The parenchyma of the liver was very moist, infiltrated with greenish bile, moderately congested, and of somewhat diminished consistence. Nothing unusual could be observed in its vascular apparatus: the circumference of the portal vein measured 4 centimètres (1⅗ English inch). Some of the hepatic cells were remarkably pale and in a great measure free from fat; others were completely filled with a bright orange-yellow substance, or with little brown masses, or with green granular pigment; only a few cells contained small drops of oil, which were not confluent. Leucine and tyrosine could only be found in small quantities in the hepatic parenchyma; sugar was not present at all.

The stomach was much contracted; its muscular tissue was covered with a grey tenacious mucus; the muscular tissue of the pylorus was thickened and penetrated by the tumour in the pancreas which pressed against it. The duodenum was covered with a thick layer of white mucus; its inner lining membrane was swollen and of a dirty-grey colour. The place where the ductus choledochus and pancreatic duct opened, projected in the form of a hard white papilla; there was no ulceration anywhere.

The head of the pancreas was firmly adherent to the wall of the duodenum, and was occupied by a grey medullary tumour, which at some places was softened. In the interior of the cancerous mass might be seen excavated spaces, resembling cysts, and corresponding to the pancreatic ducts; these contained a colourless mucus, and their walls had been eaten away by the surrounding disease. The remaining portion of the gland, which was exempt from the disease, was atrophied; the pancreatic duct was greatly distended, and furnished with numerous sacculated bulgings, shut off by valve-like folds of the lining membrane, and filled with secretion. About a drachm and a-half of fluid could be collected from this duct. The turbid fluid appeared, upon contact with the air, to become partly coagulated; the coagulum consisted of brilliant gelatinous globular masses; under the microscope there were seen corpuscles similar to those of pus, and cells which had partly undergone fatty degeneration, entangled in a grey striated matrix. The reaction was feebly alkaline; the filtered fluid obtained after trituration with water, yielded no precipitate on being boiled. Nitric acid produced a distinct turbidity, as also acetic acid; the turbidity in the latter case was partly dissipated by an excess of the acid. In the small intestine there was nothing abnormal; at the ileo-cæcal valve, the mucous membrane began to be red and swollen, and this condition was much more intense further on in the rectum. In this portion of the bowel the dark-red velvety mucous membrane was covered with a bloody viscid fluid, but there was no ulceration of the surface present. Nothing abnormal could be detected upon microscopic examination in the blood of the portal vein, or in that of the splenic or hepatic veins.

The kidneys were of normal size and of a smooth surface, and were moderately congested; their minute structure was perfectly normal, with the exception of a faint jaundiced tint of the glandular epithelium. Urine containing an abundance of bile-pigment, but no sugar, was found in the bladder.

Richard Bright had previously described a case in which diabetes manifested itself under similar circumstances to those of the present case. Observations of this nature, prove how little the formation of sugar in the liver is interfered with by a stoppage to the flow of bile. The frequency with which diabetes is accompanied by diseases of the pancreas, has appeared to me remarkable; out of nine cases I have seen atrophy or fatty degeneration of this gland in five. It is still undetermined, whether these lesions are to be regarded as the exciting causes of diabetes, and, if so, in what manner they operate.

The changes which were observed in the pons Varolii are deserving of notice, in reference to Bernard's experiment of exciting diabetes by irritating the eighth pair of nerves at their origin in the fourth ventricle; whether they stood in etiological relation to the flow of urine could not be ascertained under such a complication of circumstances.

The great influence exerted by the secretion of urine upon the intensity of the jaundice, is shown by the rapidity with which the patient became pale, from the time the kidneys began, under the operation of the sugar in the blood, to secrete freely.

Observation No. VIII.

Closure of the Ductus Choledochus by newly-formed areolar tissue, resulting from Peri-hepatitis.—Jaundice and Enlargement of the Bile ducts.—Dropsy.—Secondary Pneumonia.—Death.

C. Schmidt, aged 74, a tradesman's wife, for her age was an unusually vigorous and active-minded female, fell ill, as was stated by her ordinary medical attendant, in November, 1854. Without any obvious external cause, she was seized with symptoms which indicated the existence of gastro-intestinal catarrh:—loss of appetite, a grey-coated tongue, and diarrhœa alternating with constipation. Along with these symptoms, there was tenderness of the hepatic region, gradually increasing in intensity, so that the patient was obliged to keep her bed. These complaints lasted until the end of the year, when there was some amelioration, although only of a temporary nature. Already, in January, 1855, the patient's sufferings returned with increased intensity: the pains in the right hypochondrium were so great, that she was unable to sit up or to leave the horizontal posture. The appetite failed completely, and severe diarrhœa set in. This diarrhœa was just arrested, about the middle of February, under the use of astringents, red wine, &c., when jaundice suddenly made its appearance, and continued until death; the stools were completely deprived of colour; the skin became yellow, and by degrees, greenish-brown and bronze-coloured.

The patient was more carefully examined by Dr. Nega on the 20th of March, five weeks after the commencement of the jaundice, and shortly after, by myself. The liver lay deeper than usual; its upper margin, close to the right edge of the sternum, was found to be on a level with the seventh rib; the dimensions of the organ, were somewhat reduced in every direction. Its anterior edge, which could be easily made out on palpation, felt sharp, and a cylindrical elastic tumour, five inches long and two inches in breadth, extended beyond it. This tumour was situated at the outer margin of the rectus muscle, and was, of course, assumed to be the distended gall-bladder. Both the convex and the concave surfaces of the liver, as far as they could be felt, were smooth, and free from nodules or projections. The tenderness in the right hypochondrium was completely gone; but the digestive powers were much impaired, the tongue was covered with a greyish-white coat, there was scarcely any appetite, and there was obstinate constipation with flatulence. The pulse was 60, and never sank below this during the whole duration of the disease. The heart was normal; the lungs were emphysematous; and there was slight bronchitis, with greyish-white tenacious sputa. There was a great tendency to perspiration; severe itching had been present, but this symptom had ceased to exist. The urine was scanty, of a beer-brown colour, presenting a lively reaction with nitric acid; it was generally clear, but sometimes turbid, owing to the presence of a red sediment of uric acid.

The consciousness was unimpaired; and there never were any derangements of the senses, or yellow vision.

It was obvious, that there was here an occlusion of the ductus choledochus; the only question was, what was the cause which rendered the duct impermeable? The propagation of a chronic gastro-intestinal catarrh did not furnish a satisfactory explanation, inasmuch as a complete and persistent closure is not produced in this way. The idea that the ductus choledochus might be compressed by cancerous deposit, or by a tumour of some other nature in the liver, was entirely opposed by the smooth surfaces and the uniform sharp margin of the gland. It is seldom that an impacted gall-stone hermetically closes up the duct for a length of time; moreover, such an event is almost always preceded by attacks of colic. The tenderness in the hepatic region, which lasted for several weeks, and which was unaccompanied by jaundice, by any acute attacks of pain coming on spontaneously, or by vomiting, &c., could not be mistaken for colic; it indicated, with much greater probability, the existence of perihepatitis. Hence we assumed that it was this which had given rise to the closure of the ductus choledochus; although we did not conceal from ourselves that this supposition was only the most probable one of all others, inasmuch, as former experience had taught us,* that small tumours in the neighbourhood of the duodenum and in the head of the pancreas, can obstruct the duct without being perceptible on palpation. An unfavourable prognosis was of course arrived at; and the treatment consisted in doing little more than alleviate symptoms.

A mild, vegetable diet, with milk, and Seltzer water was ordered; extract. gratiol.† was given to keep the bowels open.

No important change in the symptoms took place during the next few weeks, except that the appetite improved, the flatulence was removed, and the stools were passed regularly, but still remained clay-coloured.

In the middle of May, œdema of the feet and ascites appeared, both of which increased with considerable rapidity, and were accompanied with anasarca of the upper half of the body; the urine was scanty and dark, but continued free from albumen. The bronchitis increased and the expectoration became more abundant and puriform;[*] and percussion indicated the presence of fluid in both pleural sacs. The appetite again fell off; and the tongue became covered afresh with a thick grey coat.

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* See Observation No. V.

† *Extractum Gratiolæ*, prepared from the herb and root of the Gratiola officinalis, or Hedge-hyssop.—TRANSL.

She was ordered the compound tincture of bark as a dietetic, and the liq. ammon. anis.[†] to promote expectoration.

A transient improvement was obtained, but still the dropsy made progress. The quantity of urine became smaller and smaller, so that fears were entertained that the uriniferous tubes had become extensively blocked up with solid deposits of pigment. An infusion of calamus root, with digitalis and acetate of potash, along with some glasses of the Karlsbad Millspring,[‡] had a beneficial effect upon the quantity and the quality of the urine; it became paler in colour; its quantity was trebled; and the dropsy was again removed.

For three weeks this favourable change continued under the use of the Millspring, when, notwithstanding a copious flow of urine, the dropsy again made its appearance; the liver became smaller, the length of the gall-bladder was gradually reduced from five to three inches, with a corresponding diminution in breadth.

In the middle of July, a pneumonic exudation took place in the right lung, extending upwards as high as the spine of the scapula. The pulse rose therewith to 90; the expectoration ceased, and afterwards became purulent; resolution took place in part of the hepatized lung, but the greater portion remained for a long time stationary, and subsequently passed into a state of suppuration. The expectoration was abundant and fetid; the patient grew more and more collapsed; the dropsy became general, until, on the 11th of August, death took place by exhaustion. Consciousness remained clear to the last. What appeared remarkable was, that throughout her entire illness the patient's temper remained unruffled; when her sufferings were in some degree bearable, she joked about her colour, felt her liver, and commissioned us to make a careful examination of it after her death.

Autopsy, 18 *hours after death.*

The skin was dark-yellow, and at some places bronze-coloured.

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[*] The sputa were always free from bile-pigment. They were frequently tested with nitric acid; but no play of colours could ever be observed.

[†] See note, page 133.

[‡] See notes, pages 58 and 124.

There was general anasarca. About twelve pounds of clear, brown fluid, were found in the abdominal cavity.

The liver extended about three centimètres (1⅕ inch) beyond the margin of the false ribs; it was somewhat reduced in size. On applying the fingers along the outer surface, soft places could be felt, varying in size from that of a pea to that of a hazel-nut, which were somewhat prominent, and, upon section, discharged a greyish-yellow fluid (enlarged bile ducts). The serous covering was at some places thickened; and the upper surface of the organ was adherent to the diaphragm by newly-formed bands of areolar tissue. The gall-bladder extended about three inches beyond the anterior margin; its breadth amounted to 1¾ inch. The walls of the gall-bladder were white and thickened from organised exudation, and its posterior surface was connected to the duodenum by cords of areolar tissue; the cystic and hepatic ducts were both enlarged to the extent of an inch in their transverse diameter. Three lines below the place where they joined to form the ductus choledochus, the canal was completely obliterated; there was here a thick, whitely-striated areolar membrane, which constricted (*lit.* tied off) the duct from without, and bound it firmly to the neighbouring duodenum. The contents of the gall-bladder consisted of a greyish-yellow mucus, in which were suspended brownish-black flakes, presenting an amorphous appearance under the microscope—the residuum of decomposed bile-pigment. The fluid yielded no distinct reaction with nitric acid. A similar fluid, but somewhat browner in colour, was found in the distended bile ducts of the liver. The distention of these ducts was, for the most part, of a uniform character; but in several places there could be observed ampulliform enlargements. The walls of the ducts were thick and rigid; they were enveloped externally by layers of white, areolar tissue, which passed inwards along with them through the hepatic fissure.

The hepatic tissue was of firm consistence, and upon its cut-section the lobules appeared of a greenish-brown colour at their peripheries, brownish-black at their centres. The distribution of the pigment was more minutely examined by making fine sections of the organ after boiling, moistening them with acetic acid, and submitting them to a magnifying power of 80 diameters. The colouring-matter could then be seen to be chiefly accumulated in the vicinity of the central veins of the lobules; the colour became gradually paler towards their circumference. Dark brown granules lay scattered throughout the parenchyma, which, when more highly magnified, were found to be hepatic cells deeply impregnated with colouring-matter. In addition to the greatly distended thick-walled bile ducts, round or elongated spaces of a brown colour were observed at many places, which I regarded as the cross sections of the smallest excretory ducts filled with stagnating bile. The hepatic cells were everywhere intact. Some of them were pale and softened, but in no way abnormal; but most of them were loaded with bile. This consisted of yellow or brown granules, either isolated or adhering in dense groups, in the interior of the cell; in a few of the cells only was a nucleus visible. Many of the cells contained abundant deposits of spherical, angular, or cylindrical little masses, of a yellow, brown, or green colour. In several, the colouring-matter was distributed in a uniform manner. Where the nucleus was visible it sometimes appeared pale, at other times tinged with green or yellow.

The deposits of bile external to the cells were of various forms and tints; most of them were cylindrical, straight, or bent, in some instances ramified, and occasionally furnished with bulging prominences; their colour was yellow, brown, ochre, or green. One would have been inclined to have taken these for casts of the most delicate of the bile ducts, had not this supposition been contradicted by the presence of similar formations in the interior of the cells. Along with these elongated forms other deposits were observed, which were rounded, club-shaped or angular. These deposits were of tolerable consistence; by compression between two glass plates, they could be split up and crushed to pieces.

Although the substance of the liver was not chemically examined for leucine, until after it had been kept for two days during the warm weather of the month of August, this substance was found in considerable quantity.

The spleen was somewhat enlarged, and presented a brownish-red colour and firm consistence. The mucous membrane of the gastro-intestinal canal was covered with a layer of grey mucus of considerable thickness; the mucous membrane itself was for the most part pale, and contained but little blood; at certain spots only was it much congested. Nothing abnormal could be detected in the duodenum.

The kidneys were of the usual size, their outer surface was smooth, and their parenchyma was of a dirty greenish-yellow colour. The epithelium of the uriniferous tubes had undergone extensive fatty degeneration, and they were for the most part, especially their nuclei, infiltrated with brown, green, or red pigment. It was only at a few spots, that copious flake-like deposits of colouring-matter could be observed; nowhere did these occur to the extent that was noticed in Case No. VI. It appeared as if these deposits had been removed, or at all events, arrested in their formation, by the use of the highly alkaline Karlsbad water.

The bronze-coloured skin was subjected to a more minute examination. The pigment was mostly contained in the deepest layer of cells of the *rete Malpighi,* which were coloured brownish-yellow, and contained dark granular deposits. The more superficial layers of cells exhibited only a pale-yellow tinge.

The sweat-glands in the axillæ were likewise deeply coloured by pigment, and contained a large number of molecules and granules, the size of cell nuclei. The cells of the adipose tissue presented a citron-yellow colour.

The thorax and skull were not allowed to be opened.*

The treatment of jaundice from obstruction is always directed chiefly against the cause of the stoppage to the flow of bile, in so far as there is any prospect of this cause being removed. Catarrh of the bile ducts and of the duodenum, gall-stones, and other mechanical obstructions will afterwards be treated in detail under the heads of each of these diseases. Where the causes are irremoveable, we must be guided in the more intense forms of jaundice by the general principles above laid down. In certain incurable organic changes in the structure of the liver, such as cirrhosis, cancer, &c., jaundice of considerable intensity is very often a subordinate symptom, which calls for no special therapeutic treatment.

II. *Jaundice without any detectable mechanical impediment to the excretion of Bile.*

The pathological origin of the forms of jaundice included under this head is less clear than that of those hitherto dwelt on. We are acquainted with only two conditions which can give rise to them—abnormal diffusion of bile, arising from some alteration in the supply of blood to the liver, and defective metamorphosis, or impaired consumption of bile in the blood. Both may be present during life, without leaving behind any certain traces of their existence in the dead body. But, even on these suppositions, it is more difficult and uncertain to form an opinion, and give an explanation of, several of the forms of jaundice belonging to this class, than is the case with the first group; we are obliged to substitute for unequivocal anatomical facts, analogies, the value of which can only be substantiated in a general way, but cannot, by any means, always be proved in detail.

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* The microscopic appearances alluded to in the above description are figured in Plate I. of the coloured Atlas.—TRANSL.

Not a trace is found here of those changes in the structure of the liver, which we meet with in the first group in a more or less marked degree, according to the seat and nature of the obstruction. The bile ducts are either empty or half filled; the hepatic cells, as under normal circumstances, are either free from colouring-matter, or only contain a small quantity; the parenchyma of the gland is at one time anæmic, pale, and soft, at another time it contains a normal amount of blood, or is congested; the contents of the bowel are found to be mixed with bile.

To this class we refer :—

A. *Jaundice from Mental Emotions.*

Physicians have at all times maintained, that jaundice can be produced by functional derangements of the nervous system. It has been thought that it might result from spasm attacking the bile ducts or the muscular tissue of the duodenum, and so producing an obstruction to the passage of the bile. Various considerations, which are opposed to such an explanation of a stoppage of bile, have already been advanced, quite independently of the fact, that the jaundice from mental emotions is developed much more rapidly than is ever the case in complete closure of the ductus choledochus. Since Claude Bernard proved, that by irritation of the fourth ventricle of the brain, the sugar formed in the liver can be made to pass off by the urine, the view,—according to which deranged innervation may give rise to jaundice,—cannot be regarded as extraordinary, although the difficulty of explaining the phenomenon is not in this way removed. So far as we at present understand the matter, derangements of the nervous functions may lead to accumulations of bile in the blood in two ways :—

a. By interruptions to the circulation of blood through the liver, arising from the influence exerted by the nerves over the caliber of the branches of the portal vein.

b. By interruptions to the heart's action, the respiratory movements, and the renal secretion.

The former of these causes would entail increased formation and absorption of bile; the latter, the existence of which appears to be indicated by the circumstances accompanying the outbreak of the jaundice, would give rise to a diminished metamorphosis of bile (in the blood), or would limit its excretion. At present, we can no more determine which of these two influences is the more powerful, than we are able to do in the case of diabetes.

In violent mental emotions induced by vexation, anger, fright, &c., the epigastrium becomes suddenly compressed, and there is dyspnœa, a feeling of suffocation, and sometimes, also, vomiting; the skin becomes pale, and soon after of a jaundiced colour, whilst large quantities of urine are secreted still devoid of colour.* In such a case, the jaundice makes its appearance in a few hours, and sometimes, as we are informed by authentic observations, in even a still shorter space of time. Villermé † mentions a case, in which two young persons quarrelled and drew their swords; one of them became suddenly yellow, and the other, terrified at this change of colour, dropped his weapon. The same writer relates the case of an abbé who became suddenly yellow, on a mad dog rushing against him. Although one may be inclined to doubt statements of this nature, still there are numerous observations which prove, that under such circumstances as those just mentioned, jaundice may be developed in a much shorter space of time than is wont to happen after the application of a ligature to the ductus choledochus. Patients, whose word cannot be doubted, often make communications of this nature, when we inquire into the previous history of their complaint.‡

Usually, this form of jaundice soon passes off, and is not attended by any further consequences. But to this there are exceptions; cases are met with in which the disease takes on a malignant character, and in which death ensues after a few days, amid severe nervous symptoms, such as delirium, convulsions, &c. Morgagni, in his thirty-seventh epistle, has related cases of this nature, and a similar case is reported by Villermé.*

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* This limited excretion of the colouring-matter in the urine is not without some influence upon the rapid increase of the jaundice of the skin.

† Dict. des Scienc. médic. Art. *Icterus*, p. 420.

‡ One must be very careful in coming to a decision in such cases. There are countries in which the popular belief that jaundice depends on mental emotions is so strong, that almost every case is attributed to that cause.

With the jaundice from mental emotions are closely connected the few observations which we possess of—

B. *Jaundice from the effects of Ether and Chloroform.*

These are interesting, inasmuch as, under the same circumstances, sugar has been observed to pass off by the urine.

C. *Jaundice from Snake-bites.*

That jaundice of an intense form may arise after the bite of venomous snakes, is a fact which was known to the ancients. Galen † relates the case of a slave who became intensely jaundiced after being stung by a viper. Mead‡ mentions similar cases, and insists particularly on the rapidity with which the yellow colour may be developed: "intra non integram horam fit flavus, quasi ejus qui ictero laborat." Under such circumstances, the colour often attains a remarkable intensity. Galen and Lanzoni § make mention of a green tinging, and Portal ‖ records the case of an apothecary known to him whose skin, in consequence of a sting from a viper, became first yellow and subsequently greenish. Results similar to those supervening upon the sting of a viper have been observed after the bites of rattlesnakes (*Moseley*), of scorpions, and of mad animals (*Bartholin*).

The older physicians attributed the production of this form of jaundice to a spasm of the bile ducts, or, as Fontana ¶ did, to a liquefaction of the bile resulting from putrid decomposition.

More detailed observations are still wanting to enable us to solve this question. That no obstruction to the flow of bile exists, is proved by the bilious character of the evacuations, both by vomiting and by stool. Still we are uncertain whether the cause of the accumulation of bile in the blood depends upon metamorphoses of a morbid character in the blood itself, or on a deranged innervation acting upon the circulation and respiration, in a manner similar to that which occurs in jaundice arising from mental emotions. In reference to this, the observations made by Cl. Bernard as to the action of Curari, are worthy of notice. The administration of this poison was found to give rise to congestion of the liver, and to the excretion of sugar in the urine.

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* See chapter on *Acute Atrophy of the Liver.*

† De locis affectis, Lib. V., cap. 8.

‡ Tentamen de vipera, p. 36.

§ Tractat. de venen., cap. V.

‖ *Op. cit.*, p. 140.

¶ Abhandlung über das Viperngift. Berlin, 1787, S. 417.

D. *Jaundice from Pyæmic Infection of the Blood.*

Maréchal[*] was the first to observe that in individuals, in whose bowels pus exists, the skin and conjunctiva, as well as the various tissues of the body, exhibit a more or less marked yellow colour. Sometimes there was associated with this symptom some anatomical change of the liver, but more frequently no such alteration existed, even when the colour was most intense; hence, the appearance was attributed to the dissemination of pus through the tissues. Since then, this yellow colour has been recognised as by no means a constant, but still a frequent, symptom of pyæmia, and it has been ascribed, at one time, to the transformation of the colouring-matter of the blood into yellow pigment, and at another time has been regarded as jaundice, dependent upon an accumulation in the blood of the colouring-matter of the bile. Bérard[†] believed that this colour could not proceed from bile, because the eyes and the urine were not tinged; and his statements, although erroneous, have been frequently repeated by recent writers. There can be no doubt that the yellow colour arises from the brown matter of the bile, and in every respect agrees with that of jaundice; unmistakable proofs of this opinion are furnished by the blood, the exudations and the urine. In general, the urine exhibits, upon testing, distinct indications of the presence of bile-pigment, as do likewise the serum of the blood, and the effusions into the serous cavities. Moreover, we can obtain from the blood the same crystalline colouring-matter as in the blood of patients who have become jaundiced from occlusion of the bile ducts.

The anatomical characters of the liver throw no light upon the mode of origin of this form of jaundice; the bile ducts are open and usually pour out a little thin secretion; the organ itself is in most cases anæmic and dry; and throughout its parenchyma products are found which indicate morbid conditions of the secretion, and of the metamorphoses of matter. To all appearances the jaundice is here the result of an impaired consumption of bile in the blood, arising from an abnormal condition of the metamorphic processes which go on in that fluid.

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[*] Recherches sur certaines altérations, qui se developpent au sein des principaux viscères à la suite des blessures ou des operations. Thèse de Paris, 1828.

[†] Dictionn. de Méd., T. XXVI., p. 491.

I annex two cases of pyæmic jaundice, the former of which is simple in its nature, but the second is interesting in many points of view.

Observation No. IX.

Contusion of the pelvic bones.—Rigors.—Somnolence.—Jaundice.—Albuminuria.—Death.—Phlebitis of the Pelvic veins.—Metastatic deposits in the lungs.—Soft anæmic liver.

Gottfried Wiesner, aged 28, on June 29th, 1854, suffered a contusion of the pelvic bones from a beam falling upon him. Retention of urine took place, which rendered it necessary to pass a catheter. Above the left pubes there was a superficial extravasation of blood; the power of progression was impaired; but no mobility of the pelvic bones could be made out.

All these complaints disappeared, and the patient wished to leave the Hospital, when, on the 8th of July, ten days after the injury, a severe rigor set in, followed by heat and some sweating. This did not return, but the patient was roused with difficulty, coughed, had some diarrhœa, and became very feverish.

On the 11th he was removed to the medical Clinique. Pulse 120. Respirations 32, and accompanied by visible movements of the scaleni muscles; speech indistinct; skin hot and dry; no pain upon pressure of the injured place, and no crepitation; stools pale and thin; urine albuminous. In the lungs extensive wheezing, and posteriorly, râles could be heard; but no dulness could be made out upon percussion. He was ordered decoction of senega root.

On the 13th, respirations 40 and panting. Pulse 120; heart's sounds clear; profuse perspiration; the conjunctiva and the angles of the mouth had assumed a yellow colour, which was rapidly increasing in intensity. Spleen enlarged; thin, involuntary stools; urine contained a quantity of bile-pigment.

On the 14th, the jaundice had increased; the pulse was irregular; drowsiness; œdematous swelling of the lower extremities.

Died at one o'clock in the following morning.

Autopsy.

Nothing abnormal in the cranial cavity.

The mucous membrane of the bronchi was vividly injected; over the surfaces of both lungs there were numerous patches of collapsed tissue, and sub-pleural ecchymoses. The pulmonary tissue was œdematous and congested; at the lower margin of the left lung, there was found a dark-red mass the size of a walnut, with a yellow softened centre; there was a similar mass in the apex of the right lung; the lower lobe of the right lung also contained numerous masses of the size of a cherry, furnished with yellow centres. On tracing the branches of the pulmonary artery, granular coagula, furnished with white points, were found in those which were nearest to the masses just mentioned; the lining membrane of the vessel was smooth, and a yellow colour could be seen through it, which was due to the presence of pus in the cellular sheath.

The heart, stomach, and intestinal canal were normal; the spleen was large and soft.

The liver was large, abnormally soft (*mürbe*), anæmic and flabby; the cells were loaded with finely-granular contents, and some of them with oil globules.

The bile in the gall-bladder was scanty, pale, and liquid.

The kidneys were soft and anæmic; the mucous membrane of the urinary bladder was covered with ecchymoses; the areolar tissue at the neck of the bladder was infiltrated with extravasated blood and gelatinous exudations; and on making a section through this part, numerous veins with thickened walls, and filled with pus, were seen on the cut surface; the hypogastric and iliac veins contained granular (*krümliche*) coagula.

The horizontal ramus of the pubes was comminuted, and the loose fragments of bone lay in an ichorous fluid; the descending ramus of the pubes on both sides was broken.

The parenchyma of the liver contained a considerable quantity of leucine; in the blood of the right ventricle there was found 1.17 per cent. of fat, rich in cholesterine; besides this, there was obtained from the same source a considerable quantity of bile-pigment deposited in the form of rods, grouped together in drusic masses.

The urine had a specific gravity of 1012, presented distinctly the reaction of the brown matter of bile, and contained small quantities of leucine.

OBSERVATION No. X.

Acute Articular Rheumatism.—Endocarditis.—Repeated Rigors.—Painful enlargement of the Spleen.—Jaundice.—Albuminuria and Hæmaturia.—Petechiæ.—Convulsions.—Coma, and Death.

Recent deposits upon the Mitral Valve.—Splenic Infarctions—Flabby anæmic liver.—Ecchymoses upon the mucous membrane of the Intestines, Bronchi, &c.

Rosina Peter, aged 24, a servant-maid, who had already been under treatment in the Hospital for incompetence of the mitral valves, was admitted on the 13th of November, 1856, with symptoms of febrile articular rheumatism. The disease had existed for eight days. The left knee and elbow were swollen and painful. Pulse 108; a loud systolic bruit, heard loudest below the nipple. The urine turbid, loaded with urates, but free from albumen; Profuse sweating. Was ordered colchicum and phosphate of soda.

About the 20th, the articular affection and the fever disappeared.

On the 22nd, at 4 in the morning, and again at 6 in the evening, there was a severe rigor, which lasted an hour, and was accompanied with great frequency of the pulse, and followed by heat and sweating. The joints remained unaffected; and there was no local pain.

On the morning of the 30th, there was a fresh rigor; the spleen was enlarged, and had become painful.

On the 2nd of December, at 6 in the evening, and on the 3rd, at 5 in the morning, there were fresh rigors; the spleen extended beyond the margin of the ribs; there was no local affection; the cardiac bruit stronger than when formerly examined.

On the 7th, great distress and uneasiness; splenic and hepatic region very painful.

On the 8th, 10th, 11th, 12th, 13th, 14th, and 16th, there were violent rigors; after this there were no real shiverings, but the pulse was very variable, fluctuating between 100 and 140 beats; there was great increase of temperature, and likewise greenish vomiting, and acute pains in the spleen. The arteries in the limbs were pervious; and the joints were normal.

On the 20th of December, slight jaundice was observed, and bile-pigment was found in the urine. She was ordered phosphoric acid.

The jaundice increased; the stools became brown; but the volume of the liver remained normal. On the 25th, shivering; pulse 136; great restlessness, impaired consciousness; in the evening, two attacks of convulsions.

On the 26th, violent convulsive fits of the entire body; loss of consciousness; pulse 140; petechiæ the size of a linseed upon the face and chest; urine bloody and turbid; in the evening death occurred.

Autopsy.

The dead body was intensely jaundiced, and covered with numerous petechiæ; upon the outer surfaces of the cerebrum and cerebellum, there lay extensive extravasations of blood, from two to three lines thick, which sank deeply into the sulci; the substance of the brain was anæmic and of normal consistence.

The mucous membrane of the bronchi was ecchymosed; both lungs were infiltrated with yellow serum, but without infarctions. There were numerous ecchymoses beneath the epicardium; the muscular tissue of the heart was very pale and abnormally soft (*mürbe*); The mitral valve was covered with a reddish-brown, dry, friable coagulum, having a thickness, at some places, of five lines; its margin was contracted and thickened; and the chordæ tendineæ adhered so as to form white cartilaginous-looking bands. The friable deposit upon the anterior flap extended into the middle of the tissue of the valve, as shown by a transverse section, and it had no well-defined margin; the intermediate tissue of the valve was loosened in its texture, partly transparent and vascular, and partly of a dirty-grey colour, with yellowish deposits scattered through it.

The spleen was remarkably enlarged, and its capsule covered at some places with recent exudation; its parenchyma was congested, and had numerous infarctions of various dates scattered through it. The most recent of these were of a reddish-brown colour; the older ones were greyish-yellow, and softened in the middle; the largest had a diameter of two and a-half inches. In one of the splenic arteries leading to a diseased spot, there was found a small plug, similar to the deposit upon the mitral valve.

The liver appeared large, pale, and soft; its colour was pale greyish-yellow, without any indication of lobules; its cells were normal, and filled with a mass of granules, and, in some instances, with oil globules. The portal vein contained fluid blood, in which there could be observed, even with the naked eye, small coagula of a brownish-black, or reddish-yellow colour; the altered condition of the colouring-matter presented by these coagula showed that they were of an old date; little masses of coagulum of a similar character were present in the splenic parenchyma, and in the blood of the splenic vein. The gall-bladder contained a considerable quantity of dark viscid bile; the bile ducts were unobstructed.

The mucous membrane of the stomach was covered with numerous ecchymoses the size of a linseed. A number of extravasated masses of blood, from one to two lines in thickness, and having an area about equal to the size of a two-thaler piece (crown-piece), lay beneath the mucous membrane of the intestine, as far as the colon; the contents of the bowel were scanty, and coloured reddish-brown.

The mesenteric glands were normal. The kidneys were covered with ecchymoses; their parenchyma was soft, and a greyish-yellow substance was infiltrated through their cortical substance. The urinary bladder contained bloody urine.

To account for the purulent infection of the blood,—the existence of which we were obliged to assume from the symptoms and course of the disease, as also from the anatomical appearances,—no lesion could be found, with the exception of the morbid changes in the mitral valve and in the spleen. Purulent and ichorous deposits and disintegrating thrombi in the interior of the veins were sought for in vain. There could be no doubt that the masses in the spleen depended upon emboli in the arteries, derived from particles which had been swept along from the deposit on the valves of the heart; it was questionable, however, whether the purulent infection of the blood arose from the same cause. Virchow * is of opinion that we cannot entirely deny that symptoms, which closely resemble those of pyæmia, may be produced by the corroding (*usurirende*) and ulcerating forms of endocarditis; and he records one case, in which he believed such an explanation to be possible; he thinks, however, that accurate observations are still wanting to corroborate the theory. Even the case before us cannot be regarded as one to the point, inasmuch as there is as much, if not greater, probability in the assumption that the purulent infection resulted from the deposits in the spleen. The coloured coagula in the portal and splenic veins showed that pathological products passed into the blood from the spleen; whether these consisted only of the decomposed red matter of the blood, or whether there were also substances which exerted a chemical action, cannot be determined. Moreover, the course of the disease showed, that the emboli in the splenic artery had existed for a long period without giving rise to any symptoms indicative of blood infection. At the commencement, previous to the rigors, the patient had no bad symptom, and only complained of pain in the enlarged spleen on the eighth day after the first rigor; not until fourteen days after this same rigor, did the train of symptoms, which are wont to accompany infection of the blood, make their first appearance.

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* Gesammelte Abhandl., S. 700 and S. 711.

The morbid changes in the heart, from which the unfavourable progress of the case originated, were partly of old, and partly of recent, date. A fresh endocarditis appeared as a complication of the acute rheumatism in the already diseased and incompetent mitral valve; this gave rise to the deposits upon the valve, and subsequently, by means of emboli, to the affection of the spleen.

E. *Jaundice from Typhus.*

Jaundice is usually a symptom of much rarer occurrence, in typhus than in pyæmia: in ileo-typhus * it is only observed in exceptional cases; but in petechial typhus † it is of frequent occurrence, several epidemics of this affection having been characterised by the frequency of jaundice. The mode of origin of jaundice under such circumstances has not yet been investigated with anything like sufficient care. In many cases, and especially in ileo-typhus, the jaundice appears to be of a catarrhal nature; usually, however, no obstruction to the excretion of bile can be detected, the existence of which is also contra-indicated during life by the fact of the stools being coloured. The liver does not present any important alteration in its texture; it is in most cases pale, soft, and shrivelled, and has its cells filled with finely-granular contents. I have repeatedly

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* The so-called Typhoid fever, or the *Fièvre typhöide* of Louis.—TRANSL.

† The true epidemic typhus.—TRANSL.

[illegible] appear [illegible] in the blood of the [illegible] or in the capillary [illegible], which appear [illegible] in a similar manner to what [illegible] in connection with pyæmia. (Observation No. [illegible]) [illegible] small coloured masses were found in the [illegible], but whether these were produced by emboli or not, could not with certainty be determined. My own opinion is, that [illegible] which accompanies the more severe forms of [illegible] typhus, must be explained in a manner similar to pyæmic [illegible].

The following observation may throw some further light upon the subject.

Observation No. III.

[illegible] Typhus—[illegible]—Albuminuria—[illegible] the [illegible]—[illegible] of the [illegible]—[illegible] in the [illegible] day.

[illegible]—[illegible]—[illegible]—[illegible] the [illegible] of the [illegible], one of the [illegible] glands.

J. M., [illegible], aged [illegible], was brought to the Hospital from the Workhouse on the [illegible] day of December, [illegible], at a time at which [illegible] were admitted from that institution thirty-one cases of typhus within a period of three days. The patient presented all the symptoms of simple typhus; [illegible]; constipation for three days; [illegible] [illegible]. He was [illegible] and [illegible].

On the [illegible] of January, [illegible], there was observed a yellow [illegible] of the [illegible] and of the [illegible] of the [illegible], which increased very [illegible]; the [illegible] was [illegible], and [illegible]; there was [illegible] in the [illegible]; [illegible] in the [illegible] of the [illegible]; the [illegible]; great [illegible]; [illegible]. [illegible].

On January [illegible], there were [illegible] of the [illegible]; the [illegible] was [illegible]; [illegible]; pulse [illegible]; a [illegible] apathetic deportment; tremors of the extremities; skin dry and dark yellow; the albumen in the urine diminished in quantity.

On the 4th, pulse 68; a thin greenish stool; towards evening, a second stool containing blood; urine paler, free from albumen; jaundice very intense; consciousness unimpaired. Was ordered to continue the muriatic acid.

On the 5th, a very bloody evacuation, which was followed by great exhaustion; pulse 96, small and weak; hepatic dulness in the mammary line 3 centimètres (1⅕ inch), and in the axillary 6 cent. (2⅖ English inches); great apathy and loss of memory.

On the 6th, pulse 88; the ecchymoses had increased in extent; bloody stools; urine pale and free from albumen; somnolence.

On the 7th, there was developed in the left parotid, a tense, painful tumour of very considerable extent, the lower part of which felt soft, and poured out a bloody serum when punctured with a needle. Pulse 96; skin dry and cool; urine brownish-black, loaded with pigment; free from albumen, but containing much urea, and traces of leucine. Slight cough with bloody sputa; no exudation in the lungs could be detected; somnolence as before; pupils normal. Was ordered benzoic acid with camphor.

On the 8th, pulse 108, very small, symptoms of pulmonary œdema; inability to expectorate; tremors of the extremities. Death ensued on the morning of the 10th, after a long agony.

Autopsy.

Brain and cerebral membranes normal. The mucous membrane of the bronchi covered with a yellow frothy serum; the lungs posteriorly hypostatic and œdematous.

The heart contained a considerable quantity of firmly-coagulated blood; its valvular apparatus and muscular tissue were normal.

Spleen small, two and a-half inches broad, and four inches long, anæmic; liver very large, and weighed 1.45 kilogr. (3.196 lbs. avoird.) pale and anæmic, of tolerably firm consistence; the cut surface presented a nutmeg appearance; the gall-bladder contained a small quantity of thin pale bile; the bile ducts unobstructed. The hepatic cells were normal, but loaded with granular contents, and some of them, infiltrated with fat. The blood of the portal vein, as also several of the capillaries of the liver contained reddish-brown, and a few black, flakes of pigment. There was no sugar in the hepatic tissue. Leucine was found in large quantity, but no tyrosine.

The mucous membrane of the stomach was of a bright-red colour; that of the intestine was pale, without any deposit in, or ulceration of, the glands; the fæcal matter in the cæcum was of a green colour; the mesenteric glands were not enlarged; in the rectum were a few hæmorrhagic erosions, the size of a linseed.

The kidneys were congested, and of a jaundiced hue; the glandular epithelium was partly in a state of fatty degeneration, and partly filled with a finely granular substance, which was rendered pale by the action of acetic acid.

Observation No. XII.

Petechial Typhus.—Jaundice.—Albuminuria.—Suppression of Urine.—Pneumonia on Right Side.—Dysentery.—Death on the seventh day.

Lardaceous Spleen of old date.—Anæmia of the Liver.—Exudation into the Right Lung.—Dysentery.—Recent exudation in the Kidneys.

C. Winzig, aged 37, a day-labourer, was admitted on the 1st of July, 1856, with the symptoms of exanthematic typhus; the patient was covered with an extensive roseolar eruption (*Roseola-eruption*), interspersed with a few petechiæ; considerable enlargement of the spleen; pulse 110.

On the 3rd, a slight jaundiced-tint of the skin was observed, and the urine contained albumen.

On the 4th, the patient was brought to the Clinique. Great muscular debility; numerous petechiæ upon the skin, which was intensely yellow; stools involuntary, very fetid, and containing blood; urine very scanty; only two ounces were drawn off by catheter in 36 hours, and the bottle, which constantly lay beside him, remained empty. Pulse 80, small and weak; respirations 10; extremites cool. Was ordered muriatic acid with spirit of nitric ether.

On the 5th, there was dulness over the lower and posterior part of the right side of the chest; feeble bronchial breathing; volume of the liver normal; spleen large; somnolence. Pulse 90; respirations 16. Collapse and cold extremities. Was ordered three grains of musk every hour.

On the morning of the 6th he died.

Autopsy, 12 *hours after death.*

The substance of the brain was somewhat congested; its consistence normal.

Bronchi pale; recent exudation and bloody infarctions in the lower lobe of the right lung.

The heart contained firmly-coagulated blood; muscular tissue and valves normal.

Œsophagus pale; the mucous membrane of the stomach exhibited bloody suffusions, but no ulceration of the surface; the mucous membrane of the jejunum and ileum was pale, that of the cæcum and colon considerably reddened, and covered with firm brown fæcal matter; in the descending colon, and reaching down into the rectum there were dysenteric deposits of an advanced stage, but no ulceration.

Spleen large and firm, and infiltrated with lardaceous matter.

Liver of normal size, of a pale-brown colour, and exhibiting here and there isolated yellowish-white anæmic patches; consistence tolerably firm. Bile ducts unobstructed; gall-bladder half-filled with a dark secretion. The hepatic cells contained pigment molecules and isolated drops of oil; lardaceous matter, such as existed in the spleen was nowhere to be seen. The chemical examination of the organ showed the presence of an unusually large quantity of sugar, besides much leucine, tyrosine, and hypoxanthine. It was very remarkable that in the spleen leucine was almost entirely absent. This substance could be detected in the thick bile, which also contained crystalline colouring-matter.

The kidneys were of a deeply jaundiced hue, considerably congested, and of soft consistence; the glandular epithelium was tinged partly greenish-yellow, and partly dark-brown; some of the tubuli contained cylindrical yellow coagula. Leucine and tyrosine were present in these organs also.

The urine collected during the last day of life contained an abundance of pigment, but no traces of albumen.

Observation No. XIII.

Abdominal Typhus; a severe rigor during convalescence; a fresh enlargement of the Spleen; great tenderness of the region of the Liver, and afterwards of the entire Abdomen.—Jaundice.—Dyspnœa.—Somnolence.—Death.

Cicatrizing Typhus ulcers in the Ileum; recent enlargement of the Spleen; round softened masses, of a brown colour, and about an inch in diameter, in the Liver.—Bile ducts unobstructed.—Peritonitis.

Carl Mauche, railway labourer, æt. 26, had been complaining since the 17th January, 1854; he was compelled to seek assistance from the Hospital, on account of painful sensations in the lower half of the right side of the chest, diarrhœa, and great weakness.

On his admission, on the 2nd of February, it was ascertained that the patient was labouring under bronchial catarrh; the pulse was 100; the tongue was moist and coated grey; the mental faculties were unimpaired; the spleen appeared to be enlarged in every direction; it extended from the level of the sixth rib downwards to four centimètres (1½ inch) beyond the margin of the eleventh rib. Infusion of ipecacuanha and gum arabic were prescribed.

On the 3rd, pulse 96. Roseola. Six thin stools, containing but little bile. Ileo-cæcal pain. No delirium.

On the 6th, pulse 120, severe cough with viscid sputa; dry, hot skin; tympanitic abdomen; more thin stools. Was ordered chlorine water.

On the 9th, pulse 88, and bisferiens; the exanthem had extended over the entire body; no motion of the bowels for two days; the urine deposited an abundance of urates; sputa bloody, without any obvious exudation in the lungs; dulness of hearing; splenic enlargement unchanged.

On the 11th, pulse 92; the eruption is fading; castor oil was administered on account of a sluggish condition of the bowels.

On the 13th, pulse 84; the dulness of hearing had ceased; the spleen was reduced in size; the skin moist, and the appetite had returned. Was ordered infusion of Peruvian bark.

On the 15th, the patient was convalescent. On the 18th, it was necessary to administer an infusion of senna, on account of obstinate constipation; this brought away solid, feculent, and afterwards pultaceous, stools.

On the 21st, the patient had a sudden violent rigor, lasting for an hour and a-half, and succeeded by an increased temperature of the skin, and frequency of pulse; the tongue was dry, and there were noises in the ears, great debility, profuse green-coloured evacuations from the bowels; the abdomen was tender and distended. Was ordered muriatic acid.

On the 23rd, pulse 136, and respirations 36; skin and conjunctiva yellow; tongue dry; abdomen tender; thin greenish stools, frequent but scanty. The spleen had again increased in volume, extending 5 centimètres (2 inches) beyond the margin of the ribs. The hepatic region was very painful on percussion, and its dulness in the mammary line amounted to 3 inches. Twelve cupping-glasses were ordered to be applied over the hepatic region.

On the 26th, the jaundiced-colour had increased; the thin, greenish stools continued, and masses of a greenish-colour were repeatedly vomited. The urine was scanty, and tinged with bile. Pulse very small, and scarcely perceptible. The tenderness in the hepatic region was increasing, and extended over the entire abdomen; great dyspnœa. On auscultation, widely extended râles were heard, but no consonating phenomena.* Somnolence. Was ordered benzoic acid with camphor, and warm cataplasms over the lower part of the abdomen.

On the 27th, death supervened under symptoms of pulmonary œdema.

Autopsy.

The membranes of the brain and the cerebral substance were considerably congested; the latter was of normal consistence. The air-passages in the upper part of the lungs were pale; in the lower part, the mucous membrane of the bronchi was reddened and relaxed; the pulmonary tissue was œdematous, at some places collapsed, but free from any solid exudation. The mucous membrane of the stomach was pale, with a few isolated ecchymoses. The serous membrane of the intestine was covered with exudation, partly recent and partly breaking down into pus. Some portions of the bowel were glued to one another and to the omentum. In the mucous membrane of the ileum a small number of typhous ulcers were found, already exhibiting a perfectly clean surface, and in process of cicatrization; no traces could be detected of recent deposits, to indicate a relapse of the typhus. The mesenteric glands were moderately enlarged. The kidneys were soft and congested.

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* Bronchophony. For an account of Skoda's theory of consonance, see Walshe on "Diseases of the Lungs and Heart," 1st Ed., 1851, p. 128.—Transl.

The spleen was remarkably hypertrophied, tense, and elastic; its tissue was soft, pultaceous, and congested; at its anterior border there was a reddish-brown infarction, the size of a walnut.

The liver was unusually soft; its surface was smooth and its margins sharp; its surfaces, on section, appeared of a reddish-brown colour; some spots were darker than the tissue immediately surrounding, and of a softer, almost pultaceous consistence. These masses were of a rounded form; measured from 1 to 1½ inch in their transverse diameter, and were separated from the surrounding more solid tissue, by a tolerably sharp line of demarcation. The bile ducts were pervious; and the gall-bladder contained a small quantity of thin pale bile.

The firmer portions of the hepatic tissue, on closer examination, presented everywhere normally-formed granular cells. These existed only in small quantity in the softened portions; here there were chiefly observed finely-granular masses, and the debris of disintegrated cells, free nuclei, oil globules, &c. The liver, after exposure to the air, yielded considerable deposits of leucine and tyrosine.

Unfortunately, the examination of the blood of the portal vein, and that of the branches of this vessel leading to the softened portions of the liver, was neglected in this case; hence, it could not be determined whether or not the blood from these sources exhibited abnormal conditions of any importance.

It is worthy of notice, that when the infectious diseases just alluded to are complicated with jaundice, a group of severe symptoms, such as hæmorrhages from the gastro-intestinal mucous membrane, &c., albuminuria, hæmaturia, suppression of the urine, &c., manifest themselves in a similar manner to what we find to be the case in yellow fever, and in the severe intermittent, remittent, and recurrent forms of fever, which are peculiarly endemic in tropical countries;—a circumstance which would appear to indicate an intimate relation between these morbid processes. The *post-mortem* appearances which we find under such circumstances, in the forms of fever more common in this country, are enlargements of the spleen and of the hepatic glands, and acute tumefactions of the liver and kidneys;* the glandular epithelium in the two last organs becomes filled with granular masses, and, at a later stage, with deposits of fat; their secretions diminish, and are sometimes suspended, whilst certain products of metamorphosis accumulate in their tissue.

In this way the liver participates, more or less actively, in the infectious blood-diseases. In typhus, its secreting function is impaired at an early period, the evacuations become pale, and upon *post-mortem* examination, a grey or greenish-yellow fluid, which, as a general rule, contains leucine is found in the gall-bladder.† The circumstances are similar in the so-called pyæmia, and its allied conditions. The formation of sugar, as well as the elaboration of bile, is diminished, and usually is soon completely arrested;‡ at the same time, substances find their way into the secretion of the gland, which, under normal conditions, or in the course of other diseases, are either absent or exist in much more sparing quantity. We find leucine, and usually also tyrosine in considerable quantity, together with a substance resembling in its characters xanthine and hypoxanthine, also a peculiar yellow substance in the form of yellow globules, and occasionally, according to Scherer, cystine. The secretion is frequently neutral, and contains ammonia. We have examined the liver, for the substances just mentioned, in a large number of diseases, and have arrived at the general result, that in typhus fever, the pyæmic or septic infection, the various exanthemata, the malignant intermittents, &c., the substances just mentioned exist in unusual quantity, whereas they are either absent or in very sparing quantity in pneumonia, tubercle, organic diseases of the heart, dysentery, diabetes, &c. It is impossible, in the present state of our knowledge of the metamorphic processes of matter, to understand all the important bearings of these facts, especially as similar products exist at the same time in other organs, such as the spleen, the lymphatic glands, and the kidneys. Still, the indications of local derangements in the metamorphosis of matter induced by the blood in the liver, and in other structures becoming infected, which the presence of these substances furnishes, are of importance. There can be no doubt that these derangements react upon the composition of the blood; the presence in the urine of leucine, the salts of valerianic acid, and sometimes, also, of substances similar to xanthine, shows that these derangements are not of a limited character, but that their products pass into the blood and the excretions. The pathological importance of these processes, however, cannot be determined with any degree of certainty, until the nature and extent of the implication in the general metamorphosis of matter of the parenchyma of the several organs and tissues, the varying character of which has only been recently investigated, is completely understood both in health and in disease.

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* Of these three organs, the spleen appears to be the one which is most frequently and most extensively implicated; then the liver; and, lastly, the kidneys. It is still uncertain whether this depends only upon the intensity of the infection, or upon different qualities of the poison. In yellow fever the affection of the kidneys is more marked and more constant than that of the spleen.

† There are cases in which a complete suspension of the secretion occurs, followed by all the consequences of acholia. See Observation No. XVIII.

‡ It is by no means always suspended. In a third of the cases of tpyhus, the liver still contains sugar, the quantity of which is for the most part small, but sometimes considerable.

In the last described group of varieties of jaundice, we have learnt to recognise this affection as a consequence of impediments to the diffusion, or to the consumption of bile, which are brought about partly by the influence of the nervous system upon the respiration, circulation and secretions, but chiefly by abnormal conditions of the processes of metamorphosis in the blood. It is only in this point of view that the affection can be regarded as of importance as a symptom, or as an indication for treatment.

The treatment has to be directed against the derangements of innervation, as well as against the abnormalities of the metamorphic processes upon which the jaundice depends. In cases where the action of these derangements is of a transient nature, as in mental affections, there is seldom any need for interference in the way of treatment; the milder antispasmodic stimulants, together with a tranquil deportment, warm baths, &c., are sufficient: when there are symptoms of danger, ether, castoreum, musk, &c., are indicated; and eventually, the measures which are recommended under the head of Acute Atrophy of the liver. In cases of snake-bite, purulent infection, typhus fever, and the allied conditions, it is the primary diseases which must be attended to; the jaundice, in itself, demands no especial treatment.

F. *Bilious Fevers and the Epidemic Forms of Jaundice.*

This is not the place to describe in detail the various forms fever which are complicated with bilious symptoms; the sole question for us to investigate here, is the mode of origin of the jaundice, which makes its appearance under such circumstances, and the influence exerted by the admixture of bile with the blood upon the course and symptoms of the primary disease; and in general terms to explain, so far as reliable materials are in our possession, the part played by the liver and its secretion in the morbid processes in question.

In ancient pathology, bilious fevers (under which name were included diseases of very diverse character)* comprised those affections in which with disturbance of the vascular system, there were associated yellow colour of the skin and conjunctiva, a bitter taste and bilious evacuations either upwards or downwards;—they were thus a very comprehensive class. The climatic and telluric agencies of the country, which formed the cradle of our science, as well as or that in which medical science was subsequently developed, furnished rich materials for observations bearing upon these diseases. They were referred by the ancients, in accordance with the tenets of a humoral pathology, to a faulty condition of the quantity or the quality of the bile, in which fluid, it was believed, the key had been found to explain diseases of the most varying nature. At the close of the seventeenth century, doubts were raised against this, as well as against other Galenic theories. Sydenham was the first who, from observations of the epidemic of 1669-70, ventured to express the view, that bilious evacuations and jaundice often constitute symptoms of only secondary importance, which may complicate diseases of very different characters. This first attempt to limit the class of bilious fevers was not followed by great results, inasmuch as the bilious constitution of the eighteenth century, brought not only the mass of physicians, but also such men as Huxham and others, back into the old beaten track. Selle and Stoll were the first to establish the fact of the participation of the mucous membranes of the stomach and intestines in the origin of these fevers; but the arbitary manner in which the latter writer attributed the various phenomena of these affections to the metastasis of bile,* was little fitted to favour further progress. Pinel expressed in a more decided manner than any of his predecessors, the opinion that the principal seat of these diseases was to be sought for in the digestive organs, and particularly in the stomach and duodenum. Broussais went a step farther, and declared that these fevers, the idiopathic nature of which he denied, resulted from inflammation of the gastro-intestinal mucous membrane, accompanied by an excessive secretion of bile. This view soon became widely extended; in Germany, chiefly on the authority of J. P. Frank, it became the custom to regard bilious fevers in general as a degeneration of gastro-enteric catarrh, and jaundice itself as of a catarrhal nature. There is no doubt that this was a one-sided view. Although there can be no question as to the independent existence of these morbid processes, as Littré has recently endeavoured to prove, still the frequency with which jaundice is met with in these fevers shows that the implication of the liver cannot be looked upon as a merely accidental complication, but that there exists between the two, a more intimate connection as regards their mutual exciting causes. The nature of this connection can only be learnt by a careful analysis of the observations hitherto made.

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* "On peut citer comme un rare modèle de confusion et de savante "obscurité la doctrine des fièvres dites bilienses." (*Pinel, Nosograph. philos.*, T. I., p. 41.)

The descriptions which have been handed down to us by the older writers, are not of much use for this purpose; and little more can be said of the accounts which have been left behind by Tissot,† Stoll,‡ Finke,§ and Pringle, because, as has already been justly observed by Rayer, very different morbid processes were grouped together, and but little attention was paid to their anatomical orgin. The best materials are to be found in the works of Annesley,‖ Boudin,¶ Haspel,** and especially of Griesinger,†† upon the diseases

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[*] Ad encephalum delata humoris biliformis portio deliria, phrenitides, apoplexias, genus omne convulsionum facit, ad fauces anginam, ad thoracem tussin, pleuritidem, &c.

[†] TISSOT, *Dissert. de febrib. bilios. anomal. seu histor. epidem. bilios.* Lausanne, 1758.

[‡] M. STOLL, *Aphorism de cognosc., &c.* 1797.

[§] L. FINKE, *De morb. bilios. anom.*

[‖] ANNESLEY, *Researches into the causes, nature, and treatment of the most prevalent diseases of India.* Vol. II., p. 419.

[¶] BOUDIN, *Traité des fièvres intermittentes.* Paris, 1842.

[**] HASPEL, *Maladies de l'Algérie,* Tom. II., p. 151.

[††] GRIESINGER, *Das biliöse Typhoid,* Archiv f. phys.Heilk. Von Vierordt.

of tropical climates, to which may be added, the writings upon yellow fever,* and a portion of the monographs upon the epidemic marsh fever of Holland in the year 1826.

Although the febrile processes which are most frequently associated with bilious symptoms differ in many respects from one another, still they have many points in common. They belong, without exception, to the group of infectious diseases (*Infectionskrankheiten*), which owe their origin to the absorption into the system of deleterious matters, of miasmata, and partly, also, as appears, of contagions. They are, for the most part, endemic in the swampy and marshy districts of hot climates, and only make their appearance in colder regions as extensive epidemics, when conditions arise particularly favourable to the development of deleterious effluvia, and to the active operation of these emanations upon the human species. They commence with changes in the composition of the blood, with which local lesions, especially of the spleen and liver, and often, also, of the kidneys, become associated. The diseases belonging to this class, when they reach an intense degree of severity, give rise to a train of symptoms having many points in common:—jaundice, hæmorrhages from the stomach and bowels, petechiæ, alarming nervous symptoms, albuminuria, suppression of urine, &c. Moreover, in most cases, the fever runs a certain course, and then suddenly ceases, either without returning (yellow fever), or followed by other paroxysms, which sometimes assume a fixed type (intermittent and remittent forms of fever), or at other times, appear as relapses without any fixed type (relapsing fever). But along with these characters in common, there exist important differences, which necessitate a rigid discrimination of the different forms.

We observe bilious symptoms as a common accompaniment of, first:—

a. Intermittent and Remittent Marsh Fevers,

and especially in those which are met with in tropical countries. The frequency with which jaundice makes its appearance under such circumstances varies at different times and places, without our being able to assign any definite cause for such being the case. In Algeria,

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1853. Heft 1 und 2. Also, *Handbuch der Spec. Path. und Therapie, redig. von Virchow.* 2 Bd. 2 Abth. "*Die Infectionskrankheiten*" of the same author

[*] La Roche, *Yellow Fever.* Philadelphia, 1855.

Boudin sometimes found this symptom present in seven-tenths of the cases of intermittent fever. In colder climates, it is only in exceptional epidemics that bilious symptoms attain a remarkable frequency, as in the epidemic which, in the year 1826, extended along the coast of the North Sea, from the Eider to the Schelde, and in the epidemic which is recorded to have prevailed in 1807 between Mende and Greifswald.

The influence which the supervention of bilious symptoms exercises over the course and terminations of fever, has not been sufficiently established by observation; in general, it does not appear to be of much consequence, because almost all the dangerous symptoms which accompany the jaundice may occur without it; it is only where the local changes in the liver attain an advanced stage that they give rise to marked results. Besides this, according to the experience of Annesley, the excessive secretion of the liver passing into the intestine, may give rise to inflammation of the mucous membrane, dysentery, &c. In addition to the slighter forms, which, under judicious treatment, rapidly subside, severe cases are met with, accompanied by typhoid symptoms and various local affections, in the course of which petechiæ appear, and hæmorrhages occur from the stomach and bowels, and in which the urine contains albumen or blood, or this secretion becomes completely suppressed. Such malignant cases bear a striking resemblance in their symptoms to yellow fever; they differ, however, essentially from that disease in their progress and in their anatomical lesions. The milder forms, for the most part, exhibit the single or double tertian type, rarely the quotidian or quartan;* the more severe cases, such as those which are accompanied by local derangements, generally appear as remittent or continued fevers.

The jaundice does not always arise in the same way; in many of the milder epidemics, the jaundice is produced by some impediment to the excretion of bile, owing to the fever being complicated with severe gastro-duodenal catarrh; in such cases, the bowels are torpid, and the stools are deficient in bile, and usually there are fundamental lesions of a more profound nature. Besides acute enlargement and softening of the spleen, which are often associated with wedge-shaped infarctions, or more frequently with abundant deposits of pigment, the liver is usually found to be enlarged from congestion and softened, its blood-vessels are seen to be filled with pigment flakes, and occasionally we observe extravasations of blood or abscesses in the parenchyma of the gland. In other cases, especially such as run a protracted course, and are accompanied by copious hæmorrhages from the stomach and bowels, the liver is found to be anæmic and jaundiced; the bile ducts are usually pervious, the gall-bladder distended, and the contents of the bowels loaded with bile. Quinine, administered in a suitable manner, exercises a favourable influence over the progress of these affections.

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* In the quartan type I have observed the jaundice to intermit: during the apyrexial period the colour became pale, and grew darker again with the return of the paroxysm.

Closely allied to the diseases just considered, as regards their anatomical lesions, and the operation of quinine, although in other respects bearing a greater resemblance to typhus, is :—

b. The Recurrent Fever.

(Das recurrirende Fieber—Relapsing Fever.—Fièvre à rechûte),

which is remarkable for the frequency with which it is accompanied by jaundice. This form of fever making its appearance in distinct, regular fits, has only recently been accurately distinguished from typhus and the intermittents, although we possess many observations of an older date as to its prevalence.* This is the fever which gave rise to the great epidemics which have prevailed in Scotland and in Ireland, and also in London, since 1843.† Bilious symptoms, such as jaundice, and bilious evacuations both upwards and downwards, occurred during the first or second paroxysm; they varied greatly in frequency; in certain of the epidemics in Scotland, they were almost of constant occurrence, so that the disease was designated "Mild Yellow Fever;" in other instances, as in London, Jenner observed these symptoms in only one-fourth of the cases, and occasionally they have been observed even less frequently. Of the anatomical lesions which in general are not very remarkable, swelling of the spleen is the most conspicuous; this not unfrequently becomes excessive, and is accompanied by the formation of infarctions.

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* See Hildenbrand, "*Ueber den ansteckenden Typhus.*" Wien, 1815. Larrey, "*Mémoires de Chirurgie militaire.*" Paris, 1812.

† Cormack, *Natural History, Pathology, &c., of the Epidemic Fever.* Edinburgh, 1843. *Dublin Journal*, 1849. Lange, *Beobachtungen am Krankenbette.* Königsberg, 1850. The author does not appear to be aware that Relapsing Fever constituted an important feature of many fever epidemics in Scotland and Ireland, before 1843.—Transl.

The liver sometimes becomes tumefied from congestion, and at other times is found soft, pale, and yellow; along with these conditions, the bile ducts are pervious, and the gall-bladder is, in most cases, filled with dark secretion. Here, as in intermittent and remittent fevers, the jaundice is, under certain circumstances, accompanied by hæmorrhages from the stomach, intestines, and other organs, by derangements of the nervous functions, lumbar pains, dysuria, retention of urine, &c., which symptoms are followed by somnolence and coma. The cause of the impaired secretion of urine has not yet been investigated with sufficient accuracy; Cormack, and other Scotch physicians, have detected urea in the serum of the cerebral ventricles and in the blood, in cases where death has been preceded by coma and convulsions, from which the existence might have been inferred, of more serious lesions of the kidneys than were found upon anatomical examination, so far as the published descriptions of the cases give us any information upon the point. This retention of urine and its consequences we shall presently find to be of much more frequent occurrence in yellow fever.

Closely connected with the recurrent fever, both as regards its progress being characterised by the occurrence of one or more paroxysms, and as to its anatomical lesions, is the fever observed by Griesinger, in Egypt, and first accurately described by him under the name of Bilious Typhoid, which, according to the experience of Lange, at Königsberg, may also be met with in the epidemic form in this country. In this fever also, morbid conditions of the spleen and of the liver constitute the most important lesions found after death; the spleen, in a few days, becomes enlarged to five or six times its normal size, its tissue is interspersed with large infarctions, and the Malpighian corpuscles not unfrequently gradually become filled with fibrinous exudation which is distintegrated into pus. The liver is at first turgid and congested; but subsequently it is found anæmic, collapsed, and jaundiced; the bile ducts are pervious, and in most cases filled with secretion. The kidneys become enlarged at an early period in a similar manner to the liver, and their glandular epithelium, like that of the liver, is filled with drops of oil. In addition to these morbid appearances, we sometimes meet with deposits in the mesenteric glands, ulcers of the larynx, such as we find in typhus, and likewise exudations, and collections of pus in various organs. Jaundice and bilious evacuations, both upwards and downwards, are common, but not constant, accompaniments of this form of fever; hæmatemesis occurs occasionally, and now and then the urine contains albumen or blood. According to Griesinger's experience, large doses of quinine exercise an unmistakeably favourable influence over the progress of bilious typhoid; but this does not appear to be always the case in the relapsing fevers of Scotland.

c. Yellow Fever.

The yellow fever bears a striking resemblance, in many of its symptoms, to bilious typhoid. Here, also, the fever ceases after two or three days, but in most cases not to return. The severe symptoms, such as hæmorrhages from the stomach and bowels, symptoms betokening a derangement of the nervous functions, lumbar pains, albuminuria, hæmaturia, and suppression of urine, going along with the jaundice, which, in the forms of fever hitherto described, have been but rarely met with, are in yellow fever regularly present; important derangements of the nervous functions also frequently manifest themselves,—for the most part, under circumstances which afford some grounds for conjecturing that they have a uræmic origin. Roche found only traces of urea in the urine;* in the blood it was detected in larger quantity. Lallemant † describes a penetrating urinous odour pervading the sweat and other secretions of patients labouring under yellow fever.

The anatomical lesions are essentially different from those of remittent and recurrent fevers, inasmuch as the swelling of the spleen, which in these affections occupy a prominent place, are usually absent in yellow fever. The liver is at first congested and swollen, but afterwards it becomes anæmic, yellow, of normal size, or somewhat smaller; the hepatic cells are found sometimes pale, in most cases devoid of nuclei, with scanty granular contents, and often filled with oil globules; the bile ducts are pervious; the gall-bladder is sometimes distended and at other times empty. The kidneys, in most cases, exhibit signs of acute exudation.

There can be no doubt that in the diseases just enumerated bilious symptoms cannot be regarded as merely accidental complications, but that they are intimately related to the fundamental causes of the maladies in question. The infection of the blood, which we regard as the starting-point of these morbid processes, first makes itself manifest by local lesions of the spleen and liver, and often of the kidneys also, which, again, in their turn, entail derangements of a special character. It is possible that these three organs mutually react upon each other, so that disease of one gives rise to disease of the others.

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* More accurate examinations of the urine in yellow fever would be of great interest, and so much the more as, in acute atrophy of the liver, very remarkable changes often take place in this secretion. (Vide *Acholia.*)

† Verbal communications.

The changes which are found in the liver afford no ground for explaining the jaundice by attributing it to catarrh of the bile ducts; the idea of a suppression of the secretion of bile is equally untenable; on the contrary, in intermittent and remittent, and also in recurrent fevers, the appearances presented by the intestinal contents and of the bile ducts is in favour of the idea of an increased secretion; or a true polycholia.* Whether these changes can be regarded as entirely the consequence of congestion of the organ, or whether the excessive formation of the products of metamorphosis in the congested and swollen spleen contributes towards them, can only be determined with certainty when the relation of the materials found in the spleen to the formation of bile has been more clearly investigated than has hitherto been possible. There are two principal causes which are to be regarded as accounting for the jaundice under the circumstances before us; first, the increased secretion of bile overloading the bile ducts, which are insufficient for its removal, and so favouring its re-absorption into the blood; and secondly, the same conditions which give rise to jaundice in typhus, pyæmia and the allied morbid processes, such as blood infection, diminished secretion of the kidneys, &c.

The anæmia and the shrinking of the liver, as also the abatement of its secretion, might be accounted for by the consumption of the blood by the splenic tumour, and partly also, by the hæmorrhage from the stomach and bowels. I cannot regard these conditions as proving the existence of an acute atrophy, in the strict meaning of that term, such as Griesinger is inclined to think may exist, because the hepatic cells remain unaffected.

Matters are somewhat different in yellow fever; in this affection, the morbid alteration of the spleen is usually absent; in general, no certain proofs exist of an excessive secretion of bile, although, on the other hand, the bilious colouring of the evacuations, and the distention of the gall-bladder with dark bile, are opposed to the idea of a suppression of this secretion, which is occasionally resorted to for explaining the existence of jaundice. The anatomical characters of the liver also are not in harmony with such a view; alterations of its intimate structure have long been sought for in vain. The congestion which is present during the first period of the disease, gives way in its subsequent progress to a state of anæmic collapse, with which is associated an infiltration of bilious matter, but the organ by no means presents the characters of acute atrophy; the hepatic cells, according to Blache remain unaffected, although they become paler, and some of them contain fat. Excepting the fundamental changes in the composition of the fluids, so far as our researches as yet extend, the abnormal distribution of the blood, arising from the relative pressure of the blood in the portal vein being diminished by the profuse hæmorrhages from the stomach, which tends to propel the bilious contents of the hepatic cells into the vascular system, alone remains to account for production of the jaundice.

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* See Annesley, *op. cit.*, Vol. I., p. 297; Vol. II., p. 429; also, Griesinger, *op. cit.*

It still remains for us to allude to the causes of the nervous symptoms, the delirium, the somnolence, and the coma, which are frequently observed in fevers complicated with jaundice.* These symptoms have at all times been accounted for by the absorption of bile into the blood; and of late the forms of jaundice associated with typhoid symptoms have been united into one group under the designation of *Icterus gravis* or *typhoides*. Whether this view is correct or incorrect, will appear by a consideration of what has been denominated cholæmic intoxication.

According to a tradition handed down from the most ancient humoral pathology, the bile, when it accumulates in the blood or undergoes certain changes, gives rise to nervous derangements of various characters, headache, delirium, convulsions, &c.† With a few interruptions, this view continued generally prevalent, even after the authority of Galen had ceased to exercise any influence. The doubts expressed by Paracelsus and Van Helmont in their day met with little sympathy, for immediately after Sylvius wrote concerning the narcotic properties of the bile, and of its capability to excite comatose fever. Boerhaave and Van Swieten[*] appealed to the testimony of the ancients in support of the dangerous effects of this secretion, and Morgagni,[†] in his description of a case of jaundice which terminated fatally, calls the bile a "materies acrior cerebrum maximé afficiens." In a similar manner, Stoll and Sarcone attributed the cause of convulsions and other dangerous symptoms to the hepatic secretion and its derivatives. In more recent times, pains have been taken to distinguish by careful examinations the various qualities of the bile, which the majority of the medical profession have during centuries believed in; but the results arrived at have been so unimportant, that for the future we must not expect that the subject will receive much elucidation from this line of investigation.[‡]

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* As regards the causes of these hæmorrhages, I would refer to the next chapter.

† "Bilis ut plurimum hominum insaniæ causa," Hippocr., edid. Kühn, III., 799. "Bilis ad caput recurrens delirii causa," Galen, Ed. Kühn, XV. 741, 598.

The attempts to ascertain more accurately the effects of the bile upon the nervous system by injecting this fluid into the blood, have been followed by such diverse results, that we are unable to draw any certain conclusions from them. It is true, that Déidier[§] found that dogs speedily died after the bile of persons labouring under the plague had been injected, or brought in contact with recent wounds; the animals also died into which Magendie[‖] injected bile; but Goupil made experiments with an opposite result, and Bouisson[¶] found that death only followed when the fluid had not previously been freed from its coarser ingredients, and when, consequently, there was nothing to prevent the capillaries of the lungs becoming plugged up. More recently, these experiments have been oftentimes repeated by Th. von Dusch,[**] but the results have been always uncertain; as a general rule, rabbits died of tetanic cramps, whereas dogs only suffered from transient indisposition and vomiting. My own experience, which is founded upon an extensive series of injections, is in favour of the complete innocuousness of an accumulation in the blood of the biliary acids and their derivatives. When bile which had been purified with the necessary amount of caution, and freed from its mucus and epithelium, or a solution of the glyco-cholate of soda, was introduced into the veins, no remarkable derangement of the nervous functions, or indeed of any function whatever, took place. The only circumstances to be observed were, that the animals immediately after the injection licked with their tongues, proving the existence of a bitter taste proceeding from the blood, and that they voided urine, which deposited a flocculent precipitate of bile-pigment, and contained the dissolved red colouring-matter of the blood, and leucine.*

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[*] Van Swieten, Comment., T. I., p. 141; T. II., p. 271; T. III., p. 499.

[†] MORGAGNI, De sedibus et causis morb., Epist. 37.

[‡] I have examined the bile in a large number of dead bodies, and have caused it to be examined in a still larger number by Dr. Valentin. In a few cases we found albumen, or sugar, and, in typhoid diseases, leucine; but, as a general rule, we could detect nothing abnormal, with the exception of varieties, in its degree of concentration, and differences in the colouring-matter, which at one time was crystalline, and at another time amorphous.

[§] De bile peste emortuorum experimenta. Halleri Bibl. anat.

[‖] Précis de physiol., T. II., p. 260.

[¶] De la bile, 1843, p. 60.

[**] Untersuchungen und Experimente, als Beitrag zur Pathol. des Icterus, &c. Leipzig, 1854.

Hence we cannot consider the accumulation of bile in the blood as the cause of typhoid symptoms, and we are under the necessity of calling in question the existence of a cholæmic intoxication.

In the fevers under consideration, this is the less likely to be the cause, as besides those forms which are accompanied with bilious symptoms, we meet with similar fevers, characterised by the same typhoid phenomena, in which jaundice is absent, and in which the bile cannot therefore be regarded as the explanatory cause of the phenomena in question. Hence we are obliged to attribute these phenomena to those obscure alterations in the composition of the blood, to which we ascribe the origin of malignant intermittents, typhous fevers, &c. Where, as in yellow fever, and occasionally also in the other forms, there is persistent suppression of urine, or where, as in bilious typhoid, there is purulent infection, the explanation of the somnolence and coma is not difficult.

The forms of fever, which we have been discussing, are for the most part peculiar to warm climates; in temperate climates, if we except the Scotch and Irish epidemics of relapsing fever, and the bilious typhoid of Königsberg, they are rarely met with.

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* Altogether, the number of injection experiments amounted to between 50 and 60, the greater number of which were undertaken, less for the object of observing the influence of the bile upon the nervous functions than in order to trace farther the transformation of the colourless bile-acids into bile-pigment. It is evident that the solution ought to be filtered before being injected; moreover, it ought not to be concentrated, because it then assumes a slimy consistence, and easily produces obstructions to the circulation. These circumstances may, perhaps, in part explain the anomalous results obtained by Von Dusch.

d. EPIDEMIC JAUNDICE.

The epidemic forms of jaundice which have been observed in Germany and France differ from the above fevers in many respects.

Among the best known are the following:—

1. The epidemic which prevailed in Essen* in the year 1772, and which was described by Brünning.† It attacked principally children, and assumed an intermittent type; various spasmodic affections and sometimes also delirium showed themselves in its course. A large number of children perished from it.

2. The epidemic in Lüdenscheid described by Kerksig,‡ ran a very mild course; less than seventy patients died of it. The jaundice usually appeared without any fever, after the precursory symptoms of gastric catarrh had lasted from eight to fourteen days; the stools were pale. Children remained completely exempt; of five pregnant women who took the disease, three aborted, two of whom were attacked on the third day after delivery with fever, which was accompanied by delirium and coma, and terminated fatally.

3. In the epidemic of Greifswald in the years 1807 and 1808, observed by Mende,§ a fourth part of all the patients had jaundice. The jaundice was in some cases unaccompanied by fever, and in other cases it was associated with fever, which was of a remittent or an intermittent character. In the latter case, it was the tertian type which predominated. During the intermission, the yellow colour not unfrequently receded, to reappear during the paroxysm; and in other cases it remained throughout the disease. One patient died under severe nervous symptoms.

4. The epidemic in Chasselay, which was observed by Chardon,‖ was very mild. The jaundice commenced with gastric catarrh, and was unaccompanied by fever; the stools were in all cases pale. Not a single case proved fatal.

5. During the epidemic which in the year 1826 prevailed along the Coast of the North West of Germany and of Holland,¶ bilious

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* Essen is a market-town in Westphalia.—TRANSL.

† De ictero spasmodico epidemico Essendiæ.

‡ Hufeland's Journ., Bd. VII.

§ Hufeland's Journ., Bd. XXXI.

‖ Bulletin de l'Acad. de Méd., 1842, T. I., p. 112.

¶ POPKEN, Historia epidem. malignæ. Jeveræ observat., 1826.

fevers frequently made their appearance along with intermittents and remittents; usually they presented a double tertian or a remittent type. Even at the present day, similar forms of fevers are occasionally met with in these marshy districts, where I have repeatedly had an opportunity of treating them.

The anatomical cause of the jaundice in the epidemics just mentioned is very imperfectly known; and hence the determination of the mode of production of this symptom must in many respects remain uncertain. The predominance of the intermittent type of fever in these cases, and the often observed increase of the jaundice during the paroxysms, are circumstances deserving of notice. In the Coast epidemic of 1826, the most constant lesions were remarkable enlargement and softening of the spleen, together with congestion of the liver,—changes the same as are found in tropical fevers, but in a milder form. After the inundation in Silesia in the year 1854, I had abundant opportunities of collecting observations on malignant intermittent and remittent fevers, some of which were complicated with bilious symptoms. Here the *post-mortem* examination, in addition to congestion of the spleen and liver, disclosed abundant accumulations of black pigment in the spleen and in the blood, as also in the other organs, especially the liver, which in most cases was softened, and had the greater number of its capillary vessels filled with this pigment. Delirium, convulsions, and coma were frequently observed during life, but the cases which were complicated with bilious symptoms, were not more remarkable in this respect than those cases in which this complication was absent.* The epidemics in Lüdenscheid and Chasselay which ran their course without fever, commenced with catarrh of the stomach and bowels, and were accompanied by light-coloured stools. They were to all appearances examples of simple catarrhal jaundice, and only differed in their extensive prevalence.

Appendix to Chapter on Jaundice.

1. *Jaundice of newly-born Children.—Icterus neonatorum.*

In a tolerably large number of newly-born children the skin and conjunctiva are tinged yellow, and the urine contains the brown colouring-matter of bile, which here, as elsewhere, indicates the existence of jaundice, and distinguishes this from all other yellow discolourations. As a general rule, the origin of this appearance is connected with changes which the function and the circulation of the liver undergo during birth. If we except the less frequent causes which may give rise to jaundice at this, as at other periods of life, such as catarrh of the bile ducts, occlusions of the ducts by thickened bile or by concretions,—instances of which have been recorded by Lieutaud, Portal, Cruveilhier, and Bouisson,* and farther, congenital obliteration of the bile ducts,† thickening of Glisson's capsule, and congenital cirrhosis of the liver,‡—we find that *icterus neonatorum* is at one time the consequence of *phlebitis umbilicalis*, but at other times is merely one of the results of the revolutions which take place in the infantile organism during birth:—a twofold origin, which the older authors had already intimated in distinguishing the more severe from the milder forms of this affection. The jaundice which is dependent upon *phlebitis umbilicalis*, is produced by the purulent infection, to which this phlebitis may give rise; it exhibits all the peculiarities of pyæmic jaundice, and terminates, as this almost invariably does, in death. The yellow tinging is here only a subordinate phenomenon in the complex train of symptoms of phlebitis.

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* See chapter VIII., on "*The Pigment-Liver.*"

The more common form of *icterus neonatorum* presents very different characters; it appears as a very mild affection, which in most cases terminates favourably, without any treatment; but which is still a subject of controversy in reference to the conditions under which it makes its appearance. It has been attributed to an accumulation of meconium (*J. P. Frank*), to catarrh of the duodenum and of the bile ducts, to spasm of the ducts, polycholia, &c.; but in most of the cases, the supposition of the existence of such exciting causes is justified as little by the symptoms during life, as by the *post-mortem* appearances.

The mode of production of *icterus neonatorum* in ordinary cases, must be sought for in the diminished tension of the capillaries in the hepatic tissue, which takes place upon the stoppage of the influx of blood from the umbilical vein, and which gives rise to an increased transfusion of bile into the blood. In strong, fully-developed infants, the equilibrium of pressure is soon restored; but in infants prematurely born, where the respiration is a long time in becoming established, and where the fœtal vessels remain long open, a more or less intense degree of jaundice is apt to make its appearance. Hence, as Bednar* and West† have rightly observed, it is infants prematurely born who are peculiarly liable to jaundice.

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* De la bile, p. 187.

† DONOP, *De ictero speciatim neonatorum Diss.* Berol., 1828. CAMPBELL, *Northern Journal of Medicine*, August, 1844. It is worthy of notice that infants succumb much earlier than adults to the effects of an interruption of the flow of bile into the intestine, and in most cases, indeed, hæmorrhages take place from the umbilical vessels.

‡ F. WEBER, *Beiträge zur path. Anat. der Neugeborenen*, III., Lief., S. 47.

Icterus neonatorum usually appears soon after birth, sometimes even within a few hours, or the colour becomes most distinct about the third day, and then, as a general rule, lasts one or two weeks. The skin and the eyes present a more or less dark jaundiced hue; the urine also is of a deeper yellow than usual, but does not exhibit the dark-brown colour of other forms of jaundice (which may be explained by its greater dilution), and often it does not even present distinctly the reaction of bile-pigment. At first, there is constipation, and the stools are pale, but afterwards they are of a normal colour; and now and then they are seen more intensely coloured than under ordinary circumstances. The general health remains unaffected; and in most cases there is no alteration in the frequency of the pulse. Certain external causes of the affection are not usually referred to; but bad nursing, and the exposure to a cold or polluted atmosphere, exercise a powerful influence, as is shown by the statistics of Foundling Hospitals.

In this simple form of *icterus neonatorum*, scarcely any treatment is required. Mild, laxative medicines, such as syrup of rhubarb, and, if necessary, a small dose of calomel with magnesia, and afterwards a few warm baths with simple diaphoretics, include all that is usually necessary under such circumstances.

2. *The Jaundice of Pregnant Females—Icterus Gravidarum.*

Pregnancy may give rise to two forms of jaundice, which in their symptoms and results present very different characters: one of these is of very little importance, but the other is associated, with serious lesion of the hepatic tissue, and almost invariably terminates in death.

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* Krankh. der Neugeborenen, IV., S. 194.

† Pathologie der Kinderkrankh. Translated from English by Wagner, S. 396. West lays great stress upon the obstructed respiration, and the impeded functions of the skin, as causes of *icterus neonatorum.*

The first of these forms makes its appearance in the later months of pregnancy, and is produced by the distended uterus, or by the accumulation of fæcal matter in the colon, exercising a degree of compression upon the bile ducts which impedes the free passage of the bile.* Sometimes the affection is developed in the earlier periods of pregnancy, and depends upon catarrh of the bile ducts, or upon mental emotions. This simple form of jaundice is attended by no consequences; it disappears upon delivery, if it has not yielded spontaneously at an earlier period to laxative medicines.†

The second form is characterised by serious derangements of the nervous system, and, so far as cases of it have as yet been examined, depends upon acute atrophy of the liver, resulting from inflammation of the parenchyma of the organ: as a general rule the kidneys are at the same time diseased. (See Chap. V. on Acholia.)

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* This view was long ago expressed by VAN SWIETEN (*Comment. ad Boerh. aphor.*, Tom. III., p. 95). VIRCHOW (*Gesammelte Abhandl.*, S. 757) observed jaundice in one pregnant female, in whom a tight-lace lobe of the liver, together with the gall-bladder, was turned up in such a way that a stoppage of bile necessarily resulted from the tension of the bile ducts.

† J. P. FRANK, however, met with a case in which a fatal rupture of the gall-bladder took place during delivery.

CHAPTER V.

SUPPRESSION OF THE FUNCTIONS OF THE LIVER.—ACHOLIA, AND ITS CONSEQUENCES.

In the last chapter, we became acquainted with a series of morbid processes, in which jaundice was in some instances accompanied by serious derangements of the nervous functions. In these cases the jaundice was associated with certain forms of fever, proceeding from an infection of the blood, and appearing as epidemics, and rarely in the sporadic form; usually no important morbid alterations of the liver were present; the cells of the gland remained unaffected, their secreting activity continued unimpaired, and frequently was even increased; the typhoid symptoms were independent either of the liver or of its secretion.

From this form of jaundice we must distinguish a second, which always comes on sporadically, and depends upon a suspension of the secretion of bile, and as a general rule is produced by acute atrophy of the liver, and in some instances by other structural diseases, such as cirrhosis, fatty degeneration, &c. It is much more dangerous than the former variety, and almost invariably terminates fatally. In their symptomatology, both have many features in common; and although extreme cases of each may present very different characters, both as regards their progress and anatomical lesions, still, on the other hand, there are cases where it is very difficult to draw a distinction, not only during life, but even on the *post-mortem* table. The principal cause of this difficulty consists in the fact, that in the first form the enlargement and the excessive secretion of the liver, are followed by an opposite condition of collapse and suppression of secretion, which in many respects resembles atrophy; a second cause is the circumstance that the anatomical diagnosis of simple typhus with jaundice is often difficult, inasmuch as the nervous derangements belonging to typhus may be ascribed to the jaundice; and a third arises from the circumstance that the suspension of the functions of the liver being far from sufficiently known, it is only provisionally inferred, that structural lesions of the liver exist. We have already seen, that enlargement and albuminous infiltration of the liver and kidneys are not unfrequently present in infectious diseases; this constitutes the commencement, or the first stage of diffuse inflammation, from which proceed destruction of the hepatic cells, and atrophy of the gland. Typhus and the allied morbid processes may in this way become the remote causes of acholia, although this condition has usually a different origin.

In order to form a clear idea of acholia, we are obliged to be guided by observations of cases, in which there has been a complete stoppage of the functions of the liver owing to an unmistakable general change in the structure of this organ. The results obtained in this manner may serve as a clue for explaining the more obscure forms.* For this purpose I have selected acute atrophy of the liver as a starting-point, and in my analysis of facts I have only made use of those observations which were undoubtedly examples of this affection.

A. THE ACUTE OR YELLOW ATROPHY OF THE LIVER.

Atrophia hepatis flava sive acuta.—Hepatitis diffusa.

1. *History and Literature.*

The first authentic communications on acute atrophy of the liver are found in Morgagni's works.† It is true, that in more ancient literature descriptions of diseases occur which correspond to this affection in their symptoms, but the anatomical proof was wanting or insufficient. Jacob Vercelloni ‡ gave an account of the disease from which his brother suffered; being hardly pressed by his creditors one night, he became suddenly jaundiced from fright, and soon fell into a restless delirium, with a remarkably irregular pulse and panting respiration, and died on the third day. Francis Rubeus * mentions an analogous case of jaundice where death occurred on the fifth day amid severe cerebral symptoms. Of a similar nature is the observation recorded by Baillou † of a boy aged 14, who, on the fifteenth day of an apparently slight attack of jaundice, with white evacuations, suddenly fell into delirium and convulsions, gave utterance to loud inarticulate sounds and died. Upon *post-mortem* examination, the brain was found healthy, "pulmo vitium insigne contraxerat, vitiosius hepar et velut ὑπερψλωρ."

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* Two mistakes have, in my opinion, been committed in treating of this difficult subject; one, that all cases of severe jaundice have been referred to acute atrophy of the liver (the old school, and especially *Horaczek*); and the other, that sufficient accuracy has not been observed in the selection of cases of typhoid jaundice, and the two groups distinguished by me have been confounded together, or, at all events, doubtful cases have been made use of in the accounts of the disease (*Ozanam* and *Lebert*).

† De sedibus et causis morborum, Epist. X., 7, et Epist. XXXVII., 2, 4.

‡ JAC. VERCELLONI, de bile aucta et imminuta, Epist. ad Bianchi, p. 194.

Bonet ‡ gave the description recorded by Guarinonius, of the history of the disease of Cardinal Sforza, who died on the sixteenth day, of an attack of jaundice, after having suffered, for the last three days of his life, from delirium and convulsions. The liver was yellow; the blood, dark and fluid. In Morgagni's works we find some of these cases repeated, and, in addition, two more instances from Valsalva's practice, in both of which the jaundice was the result of violent mental emotions. Both patients were young; and died, one, two days, and the other twenty-four hours, after the commencement of the jaundice. "Jecur inventum est flaccidum et ad subpallidum vergens."

All these observations remained for a long period dispersed through the writings of different authors, attracted but little notice, and were not referred to in any systematic work. The larger proportion of the materials in our possession have only been collected in recent times, especially since Rokitansky has accurately described the anatomical characters of acute yellow atrophy of the liver, and since Horaczek, and Budd in England, have given a general description of malignant jaundice. The bulk of the observations, however, which have been as yet accumulated, contain very heterogeneous materials resembling one another only in their external characters, so that in order to make use of them, it appears indispensable to have recourse to a careful sifting, in performing which the anatomical lesions must be our sole guide.

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* Fr. Rubeus, de ictero leth. noct. exercit., 1660, p. 195, after Ozanam.

† Ballonii Ephemerid. lib. II., p. 188. Baillou cites another similar case from Galen; who thus explains the cause of death:—"Non vi "phrenitidis, sed ob dominatum humorum virulentorum, qui sua malig-"nitate virus adæquant."

‡ Boneti, Sepulchret., p. 1007.

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II. *Symptomatology of Acute Atrophy of the Liver.*

The symptoms which accompany acute wasting of the liver are sometimes preceded by a preliminary stage; but at other times, manifest themselves directly. The precursory symptoms present nothing characteristic; usually they resemble the symptoms of an acute gastro-enteric catarrh; the patients get out of sorts, complain of dulness and headache; their tongues are coated, and the bowels are irregular, sometimes relaxed, and at other times confined; the abdomen is tender; the pulse is increased in frequency. Sooner or later, sometimes not until after the lapse of several weeks, a slight jaundiced-tint of the skin supervenes upon these derangements. The jaundice may exist in the simple form, from eight to fourteen days, or even longer, before the local changes in the liver and spleen, the hæmorrhages and the serious derangements of the nervous functions, which characterise the affection, become apparent. These symptoms, however, make their appearance about the same time as the jaundice, quite as frequently as they follow in its wake.

The disease runs a more or less violent course; in severe cases the scene terminates at the end of twelve or twenty-four hours, in other cases, after two or five days; it is scarcely ever prolonged to a week.

The symptoms usually commence with vomiting, by means of which there is thrown up first the contents of the stomach, then grey mucus, and lastly, blood, in the form of dirty-brown, or ultimately, black coffee-ground masses. At the same time, severe pains in the head come on, which, as a general rule, soon pass into delirium. In most cases, this delirium is noisy; the patients scream out and beat themselves, try to leave their beds, and are with difficulty restrained. In other instances, they are quieter, and can be roused for a short time by loud shouting. The delirium is usually succeeded by convulsions, which extend over most of the voluntary muscles, or are confined to certain localities, such as the muscles of the face and neck; now and then also they appear in the form of trismus, and in certain cases one half of the body is implicated to a greater extent than the other. Along with these spasms (which, however, are not invariably present), we observe, as a general rule, a tremulousness of the muscles of the extremities and trunk. The patient after a time becomes by degrees more and more tranquil; the state of excitement passes into stupor, and finally, into deep coma; the pupils become large, react slowly with light, and the respiration becomes sighing, intermittent, and stertorous.

The pulse, which at first was slow, and continues so as long as the jaundice remains simple, at the outbreak of the nervous symptoms increases in frequency, and gradually rises to 110 or 120 or thereabouts; at the same time, it presents remarkable variations as regards its frequency and volume: sometimes when the patients are roused, the pulse rises from 70 or 80 to 120 or 130, soon again to sink to its normal frequency or below it. These oscillations in the frequency of the pulse, which may also be observed in respect to its size and hardness, cease when the disease draws towards a close; the pulse then always increases in frequency and becomes smaller, until it can no longer be felt.

The tongue and teeth are covered at an early period with a fuliginous crust; the abdomen is in most cases, but by no means always, tender upon pressure, especially in the hypochondriac regions, and particularly the right one; even during the coma, when the hand is applied here, it produces distortions of the features, and loud complaints. The extent of the hepatic dulness diminishes more and more as the disease advances, and not unfrequently the dull space disappears completely, without there being any tympanitic distention of the bowels to account for it; at the same time the spleen is increased in volume. The bowels are almost always confined, and the stools are firm, dry, clay-like, deficient in bile, and, at a later period, not unfrequently dark coloured, and tarry from the presence of blood.

In the meantime the colour of the skin increases in intensity, and numerous extravasations of blood take place into its tissue in the form of petechiæ and more extensive ecchymoses; along with these, there are hæmorrhages from the nose, the vagina, the stomach and bowels, and the bronchi.

The urine is more or less saturated with a brown colouring-matter, presents the reaction of bile-pigment, and deposits a light precipitate, in which with the microscope there may be detected, besides amorphous mucus, the epithelium of the urinary passages, and sometimes likewise of the kidneys, coloured yellow, and also needle-shaped crystals covered with colouring-matter, either isolated or adhering in crystalline masses. Sometimes the urine contains albumen.

The various derangements just described, usually terminate after a few days in death, which usually occurs amidst increasing coma, and other symptoms of cerebral paralysis. It very seldom happens (and the observations in proof of such an event are still of a doubtful character) that, after very free evacuation of the bowels, consciousness returns, the jaundice diminishes, and recovery takes place.

III. *Illustrative Cases.*

OBSERVATION No. XIV.

Repeated attacks of Lumbago in the seventh month of Pregnancy.—Gastric Catarrh.—Icterus.—Delirium.—Convulsions.—Coma.—Death under symptoms of Blood-poisoning.

Acute Atrophy of the Liver.—Complete disintegration of the hepatic cells.—Crystalline deposits in the tissue of the liver, and in the blood of the hepatic veins.—Enlargement of the Spleen.—Abortion.

The wife of Dr. Sch., chief Physician at Kiel, 33 years of age, of short stature, delicate, and sensitive, had suffered much distress of mind during the three years' war; she often sat for an entire day in tears; she was in easy circumstances, and the mother of three children. Since her last confinement, there had been a hardness in her left mamma, which had originated under symptoms of inflammation, but subsequently had assumed the characters of scirrhus. The *post-mortem* examination showed in the sequel the carcinomatous nature of this hardness. In December, 1850, Mrs. Sch. was in the fifth month of pregnancy, and had complained of nothing save the usual symptoms incident to that condition. In consequence of an unforseen excitement, the patient was seized on rising from stool with severe pains in the muscles of the lumbar region of the spine, and was obliged to keep her bed. Although this local suffering was unimportant, still it was the commencement of a series of complaints which gradually exhausted this delicate lady, and ultimately led to a disease which terminated fatally. Fourteen days elapsed before the pains, which at the first were accompanied by smart fever, disappeared under local abstraction of blood, and rest in bed, &c. The patient remained for some days well, when on rising from stool, the pain suddenly returned at the same place, and with the same degree of violence. This time the fever was more acute, and the skin very active; the tongue was for a long time thickly coated, and there were at the same time nausea and complete anorexia. Fourteen days passed before the patient was freed from her sufferings. A second relapse, which made its appearance in the same way as the first, confined the patient to bed for a third time. Meanwhile she became emaciated; a bed-sore formed over the sacrum, which at first took a favourable course, but was very slow in healing. The patient had begun to recover, when suddenly an important change in her condition took place.

In the first place, pains were complained of in the epigastric region, and in both hypochondria, which were increased by slight pressure; the hepatic region was particularly tender. Percussion indicated a considerable diminution in the hepatic dulness, which was the more remarkable, as there was no accumulation of gas in the intestinal canal to account for it. By degrees, the dulness in the right hypochondrium completely disappeared, whilst an examination of the spleen indicated a rapid increase in the volume of this organ. The tongue again became coated; the appetite diminished; and there was repeated vomiting of a dirty-grey mucous fluid. The bowels were torpid, and had to be opened by means of senna. The stools were at first of a dark colour, and subsequently they exhibited a grey clay-like appearance. The pulse, which a short time before had been 80 to 90, sank to 64, then rose again to from 75 to 80, and towards the termination of the disease reached 110 to 130. Almost simultaneously with the pain in the hepatic region, the conjunctiva was observed to present a yellowish colour, which soon became more distinct, and extended over the skin, without, however, attaining a high degree of intensity. The cerebral functions, which, with the exception of a gloomy despondent disposition, had hitherto remained normal, underwent a sudden change, twelve hours after the outbreak of the jaundice; the patient complained of violent pains in the head, became restless, made attempts to leave her bed, and wandered in her mind. This excitement soon passed off, to make way for a rapidly-increasing somnolence. The patient lay in a stupefied state with enlarged pupils, and from time to time convulsive movements occurred of the muscles of the face, neck, and arms; she drew short sighing inspirations, each of which was succeeded by a quick expiration and a pause. The pulse was 130; the urine and stools were passed involuntarily. The skin was covered with clammy sweat. Death took place four days after the appearance of the jaundice. The treatment consisted in the administration of muriatic acid, at first by itself, but subsequently in combination with ether, and latterly with musk: purgatives, senna, and afterwards colocynth, were given along with these remedies, in order to overcome the obstinate constipation.

Post-mortem examination, 30 *hours after death.*

The body was emaciated, and of a moderately dark jaundiced hue. A fœtus, enveloped in its membranes, projected from the genital organs. The bronchial tubes and lungs were anæmic, but otherwise normal. The heart was flabby and shrivelled (*welk*); the right ventricle contained a loose reddish-brown coagulum; and the left, a little thin blood. The spleen was remarkably enlarged; its parenchyma was of a reddish-brown colour, and of normal consistence. The mucous membrane of the stomach was pale, and over the cul-de-sac and posterior wall was softened. The lining membrane of the small and large intestine was anæmic, without any marked development of its glandular system; here and there might be seen sharply-defined patches of the mucous membrane presenting a reddish-brown colour, from passive congestion; the cæcum and colon contained greyish fæcal matter.

In the sac of the peritoneum a pound of reddish-brown fluid was found, which exhibited the reaction of bile-pigment. The peritoneum itself was not injected. The kidneys were flabby and tinged yellow, and a portion of their secreting cells were in a state of fatty degeneration. The urine contained in the bladder was deeply coloured by bile; but contained not a trace of the biliary acids.* The uterus, the membranes of the ovum, the placenta, and fœtus exhibited nothing unusual. The last appeared free from any jaundiced tint, and presented by its breech. After its removal, a pound and a-half of dark, completely coagulated blood escaped from the uterus.

The liver was very considerably atrophied, flabby, and shrivelled (*welk*); it was atrophied especially as regards its thickness, which, close to the suspensory ligament, amounted to half an inch, and at the thickest part of the right lobe to scarcely one inch. The serous covering was puckered; the cut surface of the organ was smooth, shining, and of an ochre-yellow colour; there was no visible subdivision into acini. The gall-bladder contained a small quantity of grumous bile, mingled with a few small black concretions, of a neutral reaction, and exhibiting the ordinary reaction of bile-pigment and glycocholic acid. The aperture of the hepatic artery appeared to present its usual calibre, and there was nothing abnormal about its walls. The portal and hepatic veins were also of normal calibre;

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* The urine was unfortunately not tested to ascertain the amount of urea, or the presence of the other substances which since then I have found in such cases.

the bile ducts were pervious, and their lining membrane was pale. No trace of the hepatic cells could be detected upon microscopic examination of the hepatic tissue. This consisted of fine molecules, partly yellow and partly pale; and here and there were seen large, dark-brown conglomerate masses, with irregular outlines. Some drops of oil, and rounded, sharply-defined formations, which resembled the nuclei of hepatic cells, were also perceived. Amidst this detritus of the secreting apparatus of the liver, there were observed numerous needle-shaped crystals adhering together in bundles, or in radiating masses (tyrosine).*

These crystals were found in much greater abundance in the blood of the hepatic veins, which contained a thin reddish fluid, in which, along with normal blood-corpuscles, there floated numberless crystals in bundles and radiating masses.† They were entirely absent from the blood of the portal vein and hepatic artery. The liver was washed in cold water, to free it from its adherent blood; it was then cut in pieces, triturated, and boiled. The filtered fluid, upon standing, deposited numerous crystals, adhering partly in bundles, and partly in radiating masses (tyrosine). When farther concentrated, a greyish-yellow film formed at the margins and over the surface of the fluid, and there separated, a large quantity of brown globular masses, made up of concentric layers (leucine). Owing to the smallness of the portion of the liver which had been subjected to the experiment, the quantity of crystals obtained was unfortunately too minute to determine their nature with complete certainty.

OBSERVATION No. XV.

Symptoms of Gastric Catarrh and Jaundice in the seventh month of Pregnancy.—Delirium.—Convulsions and Coma.—Abortion.—Death on the seventh day of the disease.—Acute Atrophy of the Liver.—Hæmorrhage from the Intestinal Canal, and from the Mucous Membrane of the Bronchi, &c.—Peculiar composition of the Urine.

P. Nitschke, aged 24, a carpenter's wife, was brought in a semi-conscious state to the clinical department of All-Saints'. Hospital, on January 21st, 1858. The patient was a robust, well-nourished female, of a florid complexion; who, according to the statements of her relatives, had previously enjoyed uninterrupted good health; she

* Frontispiece, Fig. 1.

† Frontispiece, Fig. 2.

was in the seventh month of pregnancy. On the 17th, she had complained, as we were informed, of symptoms resembling those of acute catarrh of the stomach, loss of appetite, along with constipation, headache, general *malaise,* and great dejection of spirits, and, in short, derangements which, although not urgent, appeared sufficient to call for medical assistance. On the 20th, she came to Dr. Hasse as an out-patient, and he immediately recognising the importance of the case, directed her to be admitted into one of the medical wards. He then observed a slight yellow tinge of the face. During the ensuing night the patient, after repeated vomiting of a dirty-grey fluid, suddenly became delirious, shouted out disconnected words, and could with difficulty be kept in bed. This state of excitement still continued at the time of the patient's admission into the clinical ward; the pulse was 80; respirations 20; the temperature of the skin was not elevated; the pupils were of normal size, and acted somewhat slowly. The conjunctiva was of a pale-yellow tinge, as also the skin of the face and neck, whilst on the abdomen and lower extremities no jaundiced tinge could be recognised. The abdomen was soft, without any very great accumulation of gas; both hypochondria and the epigastrium were tender upon pressure.

On percussing the hepatic region, the dulness in the axillary line amounted to only 3 centimètres ($1\frac{1}{5}$ inch); in other situations, there was a direct transition from the sound of the intestine to that of the lung. No dulness corresponding to the spleen could be made out. The thoracic organs remained intact. She was ordered muriatic acid. In the night between the 21st and 22nd, the restlessness increased; the patient kept constantly uttering loud unmeaning cries; and the eyes remained open, the pupils being normal. The pulse rose to 112, without any elevation of the temperature, and the respirations, which were stertorous, to 26. At eleven o'clock of the 22nd, the patient was delivered of a dead seven months' fœtus, which presented no trace of jaundice. This was followed by a profuse uterine hæmorrhage. After the abortion, the excitement abated; and the patient lay, sometimes, at least, in a quiet, unconscious condition; the pulse and respiration remained unchanged; the pupils continued to act distinctly, although slowly, upon exposure to the light. The jaundice had slightly increased since the day before; the hepatic dulness was nowhere more obvious than formerly; and the spleen was equally undistinguishable by percussion.* The bowels had been confined for three days; the urine, which had to be drawn off by catheter, was acid, reddish-yellow, clear, and free from albumen; its specific gravity amounted to 1018.5. Upon the addition of nitric acid, the colour became darker, but did not exhibit the play of colours characteristic of bile-pigment. Upon standing, it deposited a slight sediment, which consisted of numerous needles, partly isolated, and partly adhering in groups, along with the yellow-tinged epithelium of the bladder and of the uriniferous tubes. Tincture of colocynth, and, subsequently, jalap, were administered along with the muriatic acid, to remove the constipation.

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* I remarked in the Clinique, that this absence of splenic dulness could

During the night, between the 22nd and 23rd, the patient lay quiet, and in a state of deep coma: muscular tremors were observed in the neck and upper extremities. The hæmorrhage from the vagina continued.

On the morning of the 23rd, the jaundice appeared increased; pulse 108; respirations 24, and stertorous; no elevation of temperature; skin dry.

From time to time vomiting occurred; by which grey mucus, mingled with brownish-black flakes, were thrown up. The bowels remained confined, notwithstanding the colocynth. The urine was darker in colour, acid, gave distinctly the reaction of bile-pigment, but not of the biliary acids; its specific gravity had risen to 1024. After standing in the cold air, a greenish-yellow, light sediment was deposited, consisting entirely of acicular crystals of tyrosine aggregated together in globular masses. When a drop of urine was evaporated upon the object-glass, it left behind a residuum, which, upon microscopic examination, was found to be almost exclusively composed of the most characteristic possible crystals of leucine and tyrosine, partially saturated with colouring-matter.* A portion of

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not be regarded as incompatible with acute atrophy of the liver, although this affection is more frequently attended by an increase of the volume of the organ, because the spleen not unfrequently lies high up in the hollow of the diaphragm in such a manner that, although enlarged, it is necessarily out of the reach of percussion, and, moreover, because old thickenings of the capsule may interfere with its enlargement.

* Plate III., Fig. 4, of the Atlas, exhibits a drop of the urine thus evaporated. The leucine was deposited partly in globular masses, with concentrically thickened walls and fissured surfaces, and partly in finely-striated laminæ: along with it there were observed greenish-yellow crystals of tyrosine. This figure has been copied in the Frontispiece of the present Translation, Fig. 5.—TRANSL.

the urine, immediately after it was drawn off by catheter, was freed from its colouring and extractive matter, by treating it with an excess of the basic acetate of lead, the excess of lead was then removed, and what remained was condensed and set aside. In twenty-four hours there was obtained a quantity of tyrosine, sufficient for several elementary analyses, in the form of brown and greenish-yellow globular crystallized masses;* the identity of which was proved, first, by the crystalline form after re-crystallization,† then by Piria's test,‡ and, lastly, by Staedeler's process of elementary analysis. The comatose condition of the patient remained unchanged: there was no enlargement of the pupils.

In the afternoon, the pulse rose rapidly to 134; the skin became covered with clammy-sweat; and death took place about 7 o'clock.

During the last two days of life, first ether, and then tincture of musk were administered along with the mineral acid.

Autopsy, 18 *hours after death.*

The body was fat, and showed no signs of incipient decomposition. The skin on the head, neck, and chest was considerably jaundiced, as also the sclerotic; on the lower extremities the colour was paler.

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* See Frontispiece, Fig. 6.

† Frontispiece, Fig. 7.

‡ In Piria's test, advantage is taken of the dark-violet colour which is produced when tyrosine is treated with sulphuric acid and perchloride of iron. If a small quantity of tyrosine, or of a substance containing a minute trace of it ($\frac{1}{20}$ grain is sufficient), be placed in a watch-glass, moistened with one or two drops of sulphuric acid, diluted, after half an hour, with water, then heated and saturated with carbonate of lime, and if to the filtered fluid perchloride of iron (containing no free acid) be added, the presence of tyrosine is indicated by the appearance of a dark violet-blue colour.

Tyrosine and leucine may be obtained as artificial products of the decomposition of albumen and fibrine, when either of these substances, but particularly the latter, is heated with dry caustic potash, or boiled with a strong solution of potash. They are also obtained in the decomposition of caseine and horn, by the action of alkalies and of acids, and by putrefaction. These substances are regarded as some of the intermediate products of the destruction of the animal tissues, between the sanguigenous bodies at one extreme, and the still less complex excreted compounds, such as uric acid, hippuric acid, urea, kreatine, &c., at the other. The composition of leucine is $C_{12}\ NH_{13}\ O_4$; that of tyrosine is $C_{18}\ NH_{11}\ O_6$. The crystalline forms presented by leucine and tyrosine are represented in the frontispiece.—TRANSL.

The skull-cap was normal; the dura mater, yellow; the pia mater, bloodless, as also the cerebral substance, which was of normal consistence; there was only a small quantity of clear serum at the base of the cranium.

The mucous membrane of the larynx, trachea, and bronchi, was of a dark-red colour from suffusion of blood; the lungs were normal, moderately congested, and posteriorly and inferiorly slightly hypostatic. The heart was of normal size, and there were numerous ecchymoses beneath the epicardium; the ventricles contained a small quantity of tar-like blood along with a little colourless coagulum. The tongue, pharynx, and œsophagus were covered with a dirty greyish-brown coat; the stomach presented a pale mucous membrane without any visible ulceration; it contained a brownish-black coffee-ground-like substance, which was also found throughout the whole of the small intestine, as far as the ileo-cæcal valve. The large intestine contained scybala of fæcal matter, but faintly tinged with bile. The mucous membrane of the bowel was everywhere pale and anæmic. Numerous ecchymoses were found in the mesentery; the veins were not enlarged nor distended; and there was no enlargement of the mesenteric glands. The spleen was fixed in the hollow of the diaphragm by old adhesions; it was enlarged by about one-third, soft, and of a pale-red colour.

The liver lay collapsed against the posterior wall of the abdominal cavity; anteriorly it was completely covered by the folds of the small and large intestine, which were displaced upwards (Fig. 10, page 49); it was dry and soft; its capsule was puckered and opaque, and its margin sharp. The dimensions of the organ were diminished in every direction, and particularly in thickness. The gall-bladder contained a small quantity of grey mucus. The tissue of the liver felt flabby and dry; in the right lobe it was at some places congested; the ramifications of the portal vein surrounding the lobules were distended, whilst the centre of the lobules presented a citron-yellow colour; here and there were small ecchymoses. A greyish-yellow substance was observed in the spaces between the lobules, which were in this case still distinctly defined, owing to the surrounding network of vessels being injected. Near the sharp margin of the liver, the capillary injection disappeared, the rounded yellow islets appeared smaller, and consequently the surrounding grey substance seemed wider. In the left lobe, where the morbid process had advanced farthest, this inter-lobular substance was still more extensive; the cut surface here presented an ochre-yellow colour, interspersed through which were distinct ramifying streaks. The secreting cells were completely disintegrated, and in their place were found numerous drops of oil and brownish-yellow molecules; it was only in the rounded border of the right lobe that a few isolated cells loaded with oil could be detected. Upon exposure the cut surface of the organ became covered with a greyish film, consisting of globules of leucine mingled here and there with crystalline masses of tyrosine. In the blood of the portal and hepatic veins the blood-corpuscles were normal, and no crystalline deposits could be detected.

The liver was injected with yellow size; this substance penetrated to the centre of the lobules, and passed thence a short distance into the capillaries, and then became extravasated, without reaching the peripheral vessels of the lobules. In fine sections of the tissue injected in this way, the enlarged central veins of the lobules could be seen surrounded by a yellow partially injected space, in which could be observed the crystalline deposits just mentioned, and this again was surrounded by a paler bluish-grey rim of densly aggregated oil globules. The weight of the liver amounted to 0.82 kilogrammes (1.807 lbs. avoird.), and the weight of the entire body to 56.2 kilogr. (123.898 lbs.), making the ratio of the former to the latter as 1 to 68.5. In healthy females of the same age and weight, the weight of the liver is about 2 kilogr. (4.409 lbs.), and the ratio is as 1 to 28. Thus the organ within six days had lost 1.18 kilogr. (2.601 lbs.) in weight. It was also considerably diminished in size; the left lobe measured 3 inches transversely, and 5¼ from before backwards; and the right lobe 5¼ inches by 5½; the thickness was 1⅛ inch.

The urine voided during life, was now subjected to a closer examination. It left behind 4.9 per cent of solid residue, and 0.14 p. c. of ash. The residuum mainly consisted of leucine, tyrosine, and a viscid extractive matter, along with traces of uric acid; urea was sought for in vain; and ammonia was present in such small quantity that the supposition of the urea having disappeared from decomposition could not be entertained. This idea was also opposed by the circumstance, that the urine examined immediately after it was voided was found to be acid. It was also remarkable, that phosphoric acid and lime were entirely absent from the ash.

Upon evaporation, the urine deposited a considerable quantity of a greenish-yellow sediment, which was collected and treated with diluted ammonia. From this solution there separated long delicate acicular crystals, possessing all the characters of tyrosine; on analysis, they yielded 8.03 per cent. of nitrogenous contents, which coincided with the formula of tyrosine. On evaporation of the ammonia, there remained behind a substance similar to tyrosine, and crystallizing in like manner, but which differed in its being more soluble, and in containing a larger proportion of nitrogenous matter. (8.83 per cent.)

The residuum of the urine was repeatedly extracted with absolute alcohol, so as to obtain any urea present. This solution upon the addition of half its volume of ether, deposited an amorphous substance, from which crystals of leucine gradually separated. The ether was evaporated from the filtrate, which was then treated with an alcoholic solution of oxalate of lime. A crystalline precipitate was formed, which was dissolved in water and decomposed by carbonate of lime. The filtered fluid when evaporated, left behind a very scanty residuum, in which, on testing with nitric acid, no trace of urea could be detected. The salt thrown down by oxalic acid consisted of oxalate of ammonia. The residuum which remained after digestion with absolute alcohol was dissolved in a great measure in boiling spirit of wine, leaving behind a viscid brown substance, in appearance and smell completely resembling that which is obtained in the formation of leucine and tyrosine from the decomposition of the proteine principles by acids. The spirituous solution when evaporated left behind a syrup, which became converted into a solid crystalline mass, from the separation of crystals of leucine.

The urine thus contained the same (or as regards the amorphous matter, perhaps only similar) substances as those which result from the artificial decomposition of the proteine compounds; whilst the urea, which, under normal circumstances, constitutes the chief product of the metamorphosis of the tissues, was wholly absent.

The blood in the heart and in the venæ cavæ contained a small quantity of leucine, and larger quantities were found in the cerebral substance, the liver and the spleen: these last organs contained a much larger amount than corresponded to what was found in the blood. Not a trace could be detected in the muscular substance of the glutæi muscles. The chemical examination of the pancreas met with an accident. Thus the liver and the spleen were the sole organs in which considerable accumulations of these substances had taken place; they are the organs which, together with the lymphatic glands and the pancreas, represent the chief formative masses of the body. The presence of a considerable amount of tyrosine in the liver, while in the spleen it could not be detected with positive certainty, favoured the supposition that this substance owed its origin in great part to the destruction of the hepatic parenchyma.

Observation No. XVI.

Jaundice in the seventh month of Pregnancy.—Severe pains in the Head.—Great restlessness.—Abortion.—Vomiting of black fluid.—Obstinate Constipation.—Coma.—Petechiæ.—Death eight days after the commencement of the Jaundice.—Acute Atrophy of the Liver.—Small Spleen.—Fatty degeneration of the Kidneys.—Abundance of Leucine and Tyrosine in the Urine.—Urea and Leucine in the Blood.

Rosalie Kienert, aged 35, was admitted on the 24th of September, 1857, into All Saints' Hospital, and died there on the 28th of the same month. The patient was a servant of robust constitution, who had previously been healthy, who, five years before, had undergone a good confinement, and who was now in the seventh month of her second pregnancy. Up to the 20th of September, she had followed her employment as a nursery maid, and had first complained during the last week of a feeling of tightness and pressure in the scrobiculus cordis. Early on the morning of the 20th, when on the point of going to her work, she was seized with a violent rigor; and at the same time violent pains in the head, loss of appetite, and jaundice of the face made their appearance, for which complaints she first sought medical aid on the 24th. On this day the patient came on foot to the Institution, in a violent fever, and scarcely in a condition to drag herself along; she blamed the treatment she had experienced at the hands of her master as the cause of her illness.

Her consciousness was unimpaired, but she was very restless, and kept constantly rolling about in bed from side to side; she suffered from violent pains in the forehead and occiput; the countenance was pale, and of a moderately yellow tinge; pupils normal; pulse 120 and soft. Vomiting occurred from time to time, by means of which undigested food, such as crude plums, was thrown up. The bowels were confined; the abdomen was greatly distended by tympanites;

no hepatic dulness could be made out; the urine was brown, and loaded with the colouring-matter of bile. Was ordered muriatic acid.

The night was spent in loud groans without sleep.

Early on the 25th, labour-pains came on, and about 1 o'clock, the patient was delivered of a six months' fœtus, which showed no traces of jaundice; during delivery there was a severe rigor and repeated vomiting of a blackish-grey grumous fluid; the patient complained of constant shivering, groaned much, and from time to time screamed out loudly.

On the 26th, there had been no sleep during the night; the consciousness was unimpaired; there was frequent vomiting of black fluid; and notwithstanding the administration of compound infusion of senna, no evacuation of the bowels had taken place. The hepatic region was tender upon firm pressure; no dulness could be made out either here or in the splenic region. Pulse small and weak, 104 to 108. Respirations 24. Great apathy.

Towards evening this apathy increased, until it merged into coma, from which the patient could not be roused. The vomiting of black fluid continued; no stool; lochial discharge scanty. There were petechiæ upon the skin of the lower extremities; pulse 104, small. The urine, which was drawn off by catheter, was free from albumen, rich in bile-pigment, and deposited upon standing, a dark reddish-brown sediment. The hepatic dulness was searched for in vain, notwithstanding that the abdomen was soft, collapsed, and painless; the vomiting of black matter continued during the patient's unconscious condition; the skin was dry and cool. Towards evening, the respiration became stertorous. At 4 o'clock on the morning of the 28th, death ensued amid symptoms of acute œdema of the lungs. During the last two days of life no internal remedies were administered, as all ingesta were immediately rejected from the stomach.

Autopsy, 12 *hours after death.*

The inner surface of the skull-cap was covered with osteophytes; and at many places was firmly adherent to the yellow-coloured dura mater. The pia mater and substance of the brain were moderately congested and normal. The mucous membrane of the bronchi was pale and covered with mucus; both lungs were very œdematous, but otherwise normal. The pericardium contained about an ounce of yellow serum; numerous ecchymoses were observed beneath the epicardium; the muscular tissue and the valvular apparatus of the heart were healthy; the cavities of the heart had their lining membrane jaundiced, and contained a considerable quantity of firmly coagulated reddish-brown blood. The mucous membrane of the œsohpagus, the epithelium of which was slightly peeled off, was covered with a black pultaceous material, which was found accumulated in large quantity in the stomach, and extended thence throughout the whole of the small intestine as far as the ileo-cæcal valve. Numerous normal blood corpuscles were detected in it by the microscope. The mucous membrane of the stomach was very relaxed, and strewn over with punctiform ecchymoses; the mucous membrane of the small intestine was pale and anæmic; solid grey fæcal matter was found in the cæcum and colon. The mesenteric glands were unaltered. The kidneys were very large and soft; their cortical substance was coloured greyish-yellow, 'and their glandular epithelium was in an advanced stage of fatty degeneration. The urinary bladder contained a large quantity of dirty-yellow acid urine free from albumen. Nothing abnormal was found in the genital organs, with the exception of the traces of the abortion.

The spleen was small, 4 inches long, 2½ broad, and ¾ thick; anæmic, and of normal consistence. Its weight amounted to 0.11 kilogr. (4 oz. avoird.)

The liver was much atrophied, and principally in its thickness; its right lobe was six inches long, and equally broad; the left, 5½ long and 3¼ broad; the thickness of the right lobe amounted to 1⅛ inch, of the left to ½. The entire weight of the organ, together with the gall-bladder and the trunks of the vessels, was 0.82 kilogr. 1.8 lb. avoird.); the weight of the body being 44.5 kilogr. (98.104 lbs.), made the relative weight of the liver to that of the body as 1 to 54.2; there was thus a diminution in the weight of the liver by nearly one-half. The consistence of the flattened gland was soft, and at some places almost pultaceous; its colour was a brownish-yellow; in the right lobe the branches of the hepatic vein were loaded with blood, which at some places appeared to be extravasated. The outer surface, especially of the left lobe, presented a wrinkled, finely-granular character, owing to the hepatic cells having disappeared at some places, and the parenchyma having collapsed; the depressed parts corresponded exactly with the circumference of the lobules. The peritoneal covering was at such places opaque and thickened. The hepatic artery appeared large; but nothing abnormal was observed in the trunk of the portal vein. Injection of the branches of the portal vein could only be made to pass along imperfectly—a remark which was still more applicable to the hepatic artery; the injected matter penetrated readily into the ramifications of the hepatic vein, but almost everywhere it avoided the tissue of the central part of the lobules, and nowhere reached the capillaries of the portal vein. On making fine sections of the tissue and drying them, its structure could be examined more accurately and traced farther. As far as the periphery of the lobules, the ramifications of the portal vein were completely filled with the injection, but the further passage of this into the capillary network was obstructed; only at a very few places was this network partially filled. The substance injected into the hepatic vein penetrated as far as the central veins of the lobules, which appeared unusually large, but only entered a short way into the capillaries in the central parts of the lobules, soon forming diffuse extravasations. A broad rim of oil globules was seen to surround the branches of the portal vein, whilst accumulations of bile-pigment, partly granular and partly diffuse, surrounded the central veins of the lobules. The hepatic cells were in some places still normal, and contained in their interior oil globules or colouring-matter; but, at other places, they were found to have become disintegrated into a finely-granular detritus. Thus the morbid process had not advanced so far as in the last case. The bile ducts were pervious, and contained but little secretion.

The urine, as well as the blood from the right side of the heart and the venæ cavæ, were subjected to a closer examination.

The urine, which had been passed twenty-four hours before death, was very acid, and deposited a thick yellowish-brown sediment, consisting, in a great measure, of urates, but containing, in addition, large yellow crystalline masses of tyrosine. The urine was further examined according to the method already described. It contained a considerable quantity of urea, and much leucine and tyrosine, together with viscid extractive matter. In this respect, the urine passed shortly before death differed from that which was collected from the bladder at the autopsy. In the latter there could only be detected an extremely minute trace of urea, and only a few crystals of nitrate of urea could be found, even with the microscope. The residuum, after evaporation of the urine, consisted of globules of leucine, intermingled with crystalline masses of tyrosine. Both substances were isolated and purified,* so that there could be no doubt as to their identity.

The blood also contained much leucine, and, what appeared important, a considerable quantity of urea. The latter was not only obtained as crystals in the form of nitrate and oxalate, but was also separated in a pure state.

In the case before us, death took place before the cessation of the local functions of the liver, and before the disintegration of the glandular cells was complete, owing to the persistent and profuse hæmorrhage from the stomach. Hence it was, that the urine secreted twenty-four hours before death still contained a tolerably large quantity of urea, whilst this had completely disappeared in Observation No. XV., in which the morbid process was more advanced. In other respects, the results of the two observations coincided. The urea which disappeared from the urine, accumulated in the blood; there was no impediment, therefore, to the formation of this substance, but only to its elimination. We see here, that without the existence of albuminuria, and without any remarkable diminution in the quantity of urine, the excretion of urea may be completely arrested. The only cause of this obstruction which could be found on the part of the kidneys was fatty degeneration of the glandular epithelium, the importance of which for the due performance of the renal functions is hereby shown; whether other causes co-operated cannot, in the present state of our knowledge, be ascertained. The quantity of urates going along with the absence of urea appeared remarkable. Can it be supposed that the conditions necessary for the secretion of urates are different from those under which urea is formed? It is obvious, that the retention in the blood of the constituents of the urine may have favoured the supervention of typhoid symptoms.

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* The detection of leucine in the urine is not unfrequently rendered difficult, from the circumstance that the extractive matters entirely prevent the formation of crystals, and the condensed extracts remain for days of the consistence of syrup. One generally succeeds in removing this impediment by digesting the extract for a long time with cold absolute alcohol, which gradually dissolves away the extractive matter: by means of boiling spirit of wine the leucine can afterwards be obtained in the crystalline form and purified. It is best to remove previously the principal portion of the extractive matter, by means of acetate of lead.

OBSERVATION No. XVII.

Symptoms of slight Catarrhal Jaundice, lasting fourteen days. On the fifteenth day, sudden maniacal delirium; hæmorrhage from the Stomach and Bowels, and Death.

Atrophy of the Liver; its secreting cells partly disintegrated, and partly in a state of fatty degeneration.

Anna Paul, aged 20, a maid-servant, of robust constitution, was admitted on October 17th, 1853. With the exception of slight attacks of cardialgia, she had always before been healthy; but for fourteen days she had complained of listlessness, loss of appetite, and tendency to constipation; along with which symptoms there was slight jaundice of the skin.

Upon admission, the epigastrium was found distended and painful; there was no enlargement of the spleen; the hepatic dulness in the mammary line measured 5 centimètres (2 inches); the tongue was dry; bowels constipated for two days. Pulse 104, small and weak; urine abundant, of a yellow colour, but without the reaction of bile-pigment. The patient was insensible, but not delirious.

She was ordered dilute muriatic acid, and an enema of compound infusion of senna.

At 7 in the evening, noisy delirium suddenly set in; the pulse was 140, and small; extremities cool. About 9 o'clock there was vomiting of black tarry matter; at 12, there were violent maniacal symptoms, and the pulse was quite imperceptible. Death took place about 5 in the morning.

Autopsy.

Brain and lungs normal; numerous ecchymoses upon the epicardium. The heart contained dark fluid blood, with gelatinous fibrinous flakes.

The mucous membrane of the stomach and small intestine was covered with black blood; the membrane itself was intact. Hard grey fæces were found in the rectum.

The spleen was slightly enlarged, soft, and congested.

The liver was small, flattened, and dry; its cut surface presented a uniform ochre-yellow colour; its consistence was at some places diminished, at other parts normal. The hepatic cells were loaded partly with fat and partly with pigment; in the softer portions of the organ they were entirely absent; here, nothing could be seen but oil globules and brown granules.

Observation No. XVIII.

Abdominal Typhus.—Profuse Epistaxis.—Violent Delirium.—Jaundice on the fifth day.—Disappearance of the hepatic dulness.—General muscular tremors.—Coma.—Death on the eighth day.

Small, shrivelled liver, with partially disintegrated cells and empty bile ducts.—Tumefaction of the Spleen.—Deposits in Peyer's patches, and in the solitary glands of the Ileum.

Gottlieb Heumann, a clerk, aged 18, came to the Hospital on November 18th, 1851, and died on the 26th.

This young man, after having felt out of sorts for some days, was seized on the 16th of November with severe rigors and heat of skin, followed by headache, general depression, and bleeding from the nose. He was ordered chlorine. On the 18th, the hæmorrhage from the nostrils returned to such an extent that plugging became necessary. Was ordered creosote. So early as the 19th, there was delirium and great restlessness; the stools were thin and pale. On the 20th, there was protracted delirium, with excitement. The spleen could be distinguished extending one inch beyond the margin of the ninth rib. Tongue dry; profuse perspiration; pulse 120.

On the 21st, slight jaundice made its appearance; the pulse was 114, and very weak; the strength was failing; the hepatic dulness in the mammary line was slight, and in the sternal line, absent; two or three thin, pale stools in the day. He was ordered muriatic acid with ether, in decoction of althæa.

On the 23rd, there was a severe rigor, lasting for two hours; great restlessness, and dyspnœa.

On the 24th and 25th, there were several attacks of tremors not unlike rigors; respirations 42; severe pulmonary catarrh; extensive rhonchi. Pulse 120, tremulous, and very weak. Stools involuntary. Was ordered benzoic acid, with camphor.

On the evening of the 25th, pulse 136; respirations 56; large drops of perspiration; complete loss of consciousness; persistent tremors of the extremities. Death on the 26th.

Autopsy on the 27th.

The abdominal cavity only could be opened.

Liver: the left lobe was visibly atrophied, with thin, sharp margins, and great diminution of its thickness; the margins of the right lobe were likewise very sharp; the entire organ was soft and shrivelled *(welk)*. The cut surface was pale-brown, completely homogeneous, with no appearance of lobules; nowhere did any coloured fluid escape from the bile ducts upon pressure; a small quantity of whitish whey-like fluid was found in the gall-bladder, the mucous membrane of which was not at all yellow. The glandular cells were in part disintegrated; some of them were very pale, and several were filled with fat.

The spleen was enlarged, very soft and dark.

The kidneys were of normal size, soft, and anæmic.

In the small intestine, there were white submucous deposits in Peyer's patches, and the solitary glands, the more extensive the nearer the ileo-cæcal valve was approached. There was no abrasion of the surface of the mucous membrane; the mesenteric glands were greatly enlarged, of a bluish-red colour, and partially infiltrated with grey deposit.

It may be a matter of question whether this case, for the accurate notes of which I am indebted to my friend Rühle, is to be regarded as one of acute atrophy of the liver, or as one of severe typhus, complicated with jaundice. The great diminution in the size of the gland, the disintegration of a portion of the hepatic cells, and the disappearance of the bile, have induced me to bring forward the case in this place. It is impossible to draw a sharp definition between the two conditions just mentioned. The diffuse infiltration of the hepatic parenchyma, and the impaired secreting functions of the organ, which, as above observed, occur not unfrequently in severe cases of typhus, are abnormal states, which differ from acute atrophy, with complete arrest of the hepatic functions, more in degree than in nature. In both cases there is an exudation, which deranges the nutrition and the functions of the gland. A case of typhus, in which the atrophy of the liver was even farther advanced, has been described by Buhl (*op. cit.*).

IV.—ANALYSIS OF THE SYMPTOMS.

In order to determine with accuracy the symptoms which arise in consequence of acute atrophy of the liver, it appears to me necessary to make a detailed analysis of the materials hitherto collected. The number of reliable observations which I have thought might without hesitation be employed for this purpose, amounts to 31. The description of some of these cases is incomplete, and is, therefore, not applicable to the determination of every question.

1. *The Premonitory Symptoms.*

These were described in one half of the cases, and usually consisted of those derangements which are met with in acute catarrh of the stomach and bowels, occasionally of rheumatic affections; upon these symptoms jaundice supervened, which, in its characters, was in no way distinguishable from simple jaundice until the symptoms of impending danger suddenly broke out. In most cases, the duration of these premonitory symptoms amounted to from three to five days; but in many cases, to from two to three weeks and upwards.

2. *The Skin.*

This was invariably jaundiced, although the colour seldom attained a high degree of intensity. So far as my own observations extend, this colour did not appear at all conspicuous. As a general rule, it commenced in the upper half of the body, on the face and neck; it extended over the lower extremities less frequently, and was here scarcely observable. Along with the colour, the skin was usually cool, dry, and inactive; in none of my own cases was any elevation of temperature observed. Alison and Bright likewise make particular mention of this circumstance. It is only at the commencement, during the premonitory febrile stage, and afterwards for a short period in the stage of great nervous excitement, during the restless delirium and convulsions, that the temperature has been observed to be elevated.*

In the advanced stage of the disease, the cutaneous system not unfrequently participates in the hæmorrhages which proceed from various parts of the body, and especially from the mucous membranes; petechiæ and large ecchymoses, of a reddish-brown or black hue, become developed. These are by no means of constant occurrence, being wanting in two-thirds of the cases.

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* Wunderlich (*Handb. der Pathol. und Therapie*, 2*te. Aufl.*, Bd. IV., S. 655) states, that he has observed in several cases of malignant jaundice a sudden rise from the normal temperature up to 34° cent. (99.2° Fahr.): there is no statement, however, to show what form of malignant jaundice he alluded to.

3. *The Organs of Circulation.*

The heart's action presents great variations in acute atrophy of the liver. If there are premonitory febrile symptoms, the pulse, upon the appearance of the yellow colour, becomes less frequent, and remains in this condition as long as the jaundice retains a simple character. Not until the abnormal nervous symptoms become prominent does the rapidity of the pulse begin to increase, and it then rises from 50 or 60 to 90 or 100, or upwards. Remarkable variations in its frequency are at the same time observable; for a brief period it rises to 110, 120, or 130, soon afterwards to fall again to 80 or 90. This fluctuation, which is equally observed in reference to the size and hardness of the pulse, may be repeated several times in the course of a few hours. Not until incipient cerebral paralysis supervenes does the increased frequency continue of a uniform character; then it sometimes reaches 140 to 150 beats, whilst its character is small, thready, and intermittent.*

To these abnormal conditions of the cardiac and vascular functions are not unfrequently superadded hæmorrhages, which usually take place simultaneously from various parts of the body; they were observed in one half of the cases. They were met with most frequently from the mucous membrane of the stomach and bowels, in the form of hæmatemesis and melæna (ten times). Uterine hæmorrhages were equally common; they were especially observed in the case of pregnant females, and then most frequently induced abortion. Petechiæ, large ecchymoses of the skin, and epistaxis were of less frequent occurrence; renal hæmorrhage was only present in exceptional cases (*Buhl*). Other hæmorrhages were almost always found in the dead body, which it was impossible to recognise during life. Among these may be especially mentioned those which took place from the portal system, extravasations between the folds of the mesentery, in the omentum, upon the serous surface of the bowel, hæmorrhagic infarctions of the spleen, and likewise, although less frequent, extravasations beneath the pleuræ and the pericardium, and upon the mucous membrane of the pharynx and bronchi, hæmorrhagic infarctions of the lungs, &c.

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* Even the earlier observers were astonished at this variable condition of the pulse. Vercelloni (*Bianchi*, *loc. cit.*, II., 794) speaks of it as "pulsus inæqualis tum quoad robur, tum quod numerum vibrationum."

4. *The Respiration.*

The respiration remains unaffected during the first stage of the disease; the mechanism of the respiratory movements is unaltered, and their frequency bears a normal ratio to that of the pulse, or is only slightly at variance with it. It is only in the later stages, when the oscillations in the heart's action, just mentioned, commence, that the respiratory motions, at least in many cases, are wont to be abnormal, and the breathing becomes sighing or stertorous. In such cases, a short inspiration, accompanied by a groaning noise, is followed by a rapid expiration, and then there is a long pause, similar to what is observed in animals in which the pneumo-gastric nerves have been divided. Structural changes of the air-passages, capable of impairing the functions of the lungs, seldom existed in the cases recorded, and it was only in exceptional instances that there were found hæmorrhagic infarctions and sub-pleural extravasations accompanying hæmorrhages in other parts of the body.

5. *The Organs of Digestion.*

These always undergo important functional changes. The premonitory symptoms usually are ushered in by gastric derangements, loss of appetite, oppression in the prœcordium, nausea, furred tongue, constipation, &c.; when there is no premonitory stage, these symptoms usually make their appearance on the outbreak of the jaundice. Among the most important symptoms belonging to this group may be mentioned, in the first place, abdominal pains, which were observed in three-fourths of the cases. They are situated sometimes in the epigastrium; at other times, and most frequently, in the hypochondria, especially the right, corresponding to the region of the liver. Pressure in this place gives rise to marked indication of pain, which is even observable during coma, from the distortion of the countenance. The pains also come on spontaneously; and the patients complain, especially during the first stage of the malady, of an uneasy sensation in the cardiac region. Although the abdominal walls are very tense and tender, there is usually no fulness present in the hypochondria.

The results of percussion are of much greater diagnostic importance than the pain, which in many cases is completely absent. Percussion shows a rapid diminution in the volume of the liver, commencing with the left lobe and extending towards the right. Usually the hepatic dulness soon disappears entirely, because the organ, constantly becoming softer, owing to the disease of its tissue, collapses, and is pushed towards the vertebral column by the intestines distended with gas. In the same proportion as the liver is atrophied the spleen is enlarged, and is the seat of tenderness, on pressure, in the left hypochondrium. It is only in rare cases that the increase in volume of the spleen cannot be made out, owing to the organ being inaccessible to percussion, from old adhesions fixing it in the hollow of the diaphragm, or owing to there being no enlargement at all. The latter may be the case when there is thickening of the splenic capsule, which prevents the organ enlarging, or when there is profuse hæmorrhage from the stomach and bowels to such an extent as to drain the portal system.

Along with these changes in the volume of the liver and spleen, which, with some care, are easily recognised, repeated vomiting is usually observed at an early period. The vomited matters have in most cases contained at first grey mucus, and occasionally bilious matter, but after a time they have consisted of a greyish-brown or black grumous fluid, the colour of which is more or less dark, according to the severity of the gastric hæmorrhage. When the hæmorrhage is slight, I have noticed, mingled with the mucous fluid, numerous brown flakes, consisting of decomposed blood, which Morgagni observed long ago, and designated as a "*materies subobscurus.*"

Almost invariably there has been obstinate constipation, which could only be overcome by strong purgatives; the stools have been dry and clay-coloured; and, at a later period, they have assumed in many cases a dark tarry aspect, in consequence of intestinal hæmorrhage.

6. *The Urinary Organs.*

The urine according to my observations, has always been secreted in normal quantity; but in the more advanced stages of the affection it has been necessary to draw it off by the catheter, for the purpose of examination, otherwise, it has passed off involuntarily. It was always of acid reaction; its specific gravity varied from 1012 to

1024; at first it only indistinctly presented the reaction of bile-pigment, and it was not until a later period that this could be detected with certainty. The remarkable changes in the composition of the urine, the appearance in large quantities of leucine, tyrosine and extractive matters of a peculiar nature, together with the gradual disappearance of the urea and phosphate of lime, as was found to be the case in Observations XV. and XVI., are conditions which hitherto have been found in no other disease. These properties of the urine indicate the existence of deeply important, although long unrecognised, abnormal states, of the metamorphosis of matter, and they furnish (provided further observation shall, as I have no doubt, show them to be of constant occurrence), no small insight into the transformations which take place in the albuminous principles, in cases where the functions of the liver are arrested. The peculiarity of the urine of most value in a clinical point of view is the deposit upon exposure to the cold, of a greenish-yellow precipitate, which, even with the naked eye, and still more readily upon microscopic examination, can be recognised as differing from all other deposits; to this may be added, the appearances presented by a drop of the dried urine. (Vide *Frontispiece.*) Further evidence can only be obtained by careful chemical analysis.

It ought also to be mentioned, that occasionally small quantities of albumen have been found for a short period in the urine.

7. *The Nervous System.*

Abnormal conditions of the nervous system have been observed in every case as essential, characteristic symptoms; and they have almost invariably presented the same general characters, although in some cases there have been sundry marked deviations. In most cases we can distinguish two stages, that of excitement, and that of depression, the former of which is characterised by delirium and convulsions, and the latter by a progressively increasing coma, gradually verging into cerebral paralysis. Only in rare cases (in one-sixth of the whole number), was the stage of excitement absent, the patients falling at once into a state of typhoid prostration, which passed into unconsciousness, somnolence, and finally, into coma.

The nervous derangements are ushered in with severe headache, accompanied by a gloomy, irritable temper and restlessness. These symptoms are soon followed by delirium, which is usually noisy, and raging, but occasionally shows itself in the form of quiet, harmless wandering. The patients throw themselves about in a restless manner, moan loudly, and from time to time utter an inarticulate cry; not unfrequently they fall into maniacal paroxysms.*

Convulsions accompanied the delirium in ten cases, or in one-third of the total number of observations; in some instances they extended over the entire muscular system, like those of epilepsy, and not unfrequently commenced like these with a shrill cry; in other cases again, they consisted of general muscular tremors, resembling a rigor; while, lastly, in some cases they appeared as partial twitchings of the muscles of the face, neck, or extremities, or in the form of hiccup, grating of the teeth, &c. Trismus, and even sometimes tetanic spasms, have been observed in a few cases.

Towards the termination of the disease, the delirium and convulsions, as a general rule, have given place to stupor, which in a short time has merged into the deepest coma, from which no shouting nor shaking could rouse the patient. The characters of the pupils have been by no means constant; in many cases they remained of normal size, and reacted with light, as in my own observations, and in those of Frey, &c.; in other cases again, they were dilated and immoveable, and in very rare instances, contracted. Yellow vision occurred in exceptional cases only.

In most cases, the nervous derangements appeared simultaneously with the jaundice; and they usually attracted the attention of the observer sooner than the slight jaundiced-tint of the conjunctiva, and of the skin surrounding the alæ of the nose. Sometimes matters were different, the jaundice lasting 2, 5, 8, 14, 17, or even 21 days, without any impairment of the nervous centres, until suddenly, the scene became transformed, and a train of symptoms betokening danger supervened.

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* This character of the delirium in jaundice appears to have been known to the ancients. Hippocrates (*ad Democritum philos. epist.*) observes:— "Qui ex pituita insaniunt quieti sunt, qui vero ex bile hi verberant, male-"fici sunt, neque quiescunt." Hippocrates further observes (*De morbo sacro*):—"Qui ex bile insaniunt clamosi, maligni et minime quieti sunt, "semper aliquid intempestivum faciunt." Ballonius also (*Epidem. ephemer.*, lib. II., cap. 188) describes the delirium as "vox inarticulata, ejulatus "magnus."

V. DURATION AND MODE OF TERMINATION.

The symptoms just described usually run their course in a few days; in most cases the entire process terminates within the first week, but occasionally the disease is protracted to three or even four weeks. In such cases it is the apparently simple jaundice of the preliminary stage which is protracted. After the commencement of the characteristic symptoms, the disease almost invariably terminates in five days, and sometimes even in from 12 to 36 hours. In 3 of the 31 cases, the date of the commencement was unknown; in the remaining 28 cases, the fatal termination occurred:—

during the 1st week	....	13 times.
„ 2nd „	....	6 „
„ 3rd „	....	5 „
„ 4th „	....	4 „

The termination has almost invariably been fatal; this result has been so constant, that the few observations which have been recorded of a cure having taken place, must be regarded as of a very doubtful character, and the more so, as most of them have occurred at a time when only a few examples of the disease had been collected, and when there was nowhere to be found any accurate account of it to confirm the diagnosis. Griffin* mentions two cases, and Hanlon† one, which terminated favourably; Budd also makes mention of one case of cure, which took place notwithstanding the previous occurrence of bloody stools, pains in the hypochondria, hiccup, and coma. In the year 1854, I treated successfully a lady, aged 40, for jaundice, associated with symptoms which favoured the supposition that there existed incipient atrophy of the liver; still I would not venture to assert that this condition was really present. In this case, the jaundice was slight, and was accompanied by delirium and typhoid somnolence, the right hypochondrium was painful; the hepatic dulness was diminished and, in the epigastrium, had completely disappeared; the spleen was enlarged. The pulse varied between 88 and 104; the bowels were confined and the stools pale; there were repeated attacks of epistaxis, &c. After eight days, these symptoms ceased, and the patient slowly recovered. The treatment employed consisted in drastic purgatives and mineral acids.

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* *Dublin Med. Journ.*, 1834, IV., 12.

† Graves, Clinical Medicine. 2 ed., 1848, Vol. II., p. 255

When the anatomical lesions, from which this disease is thought to originate, have made considerable progress, and the larger portion of the hepatic cells have become disintegrated, one can understand that a cure can no longer be thought of.

The prognosis is thus, under all circumstances, in the highest degree unfavourable.

VI. ANATOMICAL LESIONS.

The structural changes, which we find on *post-mortem* examination to account for the derangements of the various functions just described, are very diverse; the only constant ones are lesions of the liver, next in order to which come those of the spleen. All other pathological states are inconstant; they may be absent without the symptoms during life undergoing any important alteration. Hence we regard the liver as the peculiar seat of the disease, from which the derangements in the functions of other organs take their origin. The liver was in all 31 cases considerably atrophied; the diminution in volume was estimated at one-third, one-half, or even two-thirds of the normal size. Accurate measurements and weights were, however, only made in a few instances. Bright saw the weight reduced to 2 pounds, to 23 ounces, and in a third case, to 19 ounces: I have myself in two instances, found the liver to weigh only 0.82 kilog. (1 lb. 13 oz. avoird.) the relative weight to that of the entire body being as 1 to 68.5, and as 1 to 54.2, which indicated a reduction of more than one-half.

The size of the organ is diminished in every direction, but especially in its thickness; the gland is flattened out. The capsule presents an opaque puckered appearance; the parenchyma is flabby and shrivelled *(welk)*, so that it is unable to bear its own weight, and folds up and collapses in front of the vertebral column.

The cut surface of the organ presents at those places where the disease has advanced farthest, and these are usually in the left lobe, an ochre-yellow or rhubarb-like colour; the blood-vessels are here empty, and in most cases the outline of the lobules is no longer visible. At other places, where the morbid process is at an earlier stage, some of the capillaries are filled with blood, and occasionally there may be seen extravasations or their remains in the form of crystals of hæmatoidine. Between the lobules, which are encircled by the congested vessels, and separating them from one another, a dirty greyish-yellow substance is deposited. At a later period the capillary congestion recedes, the size of the lobules diminishes, and their colour becomes yellower, whilst the relative amount of the intervening grey substance gradually increases. After a time, this grey substance disappears at those places where the atrophy of the gland is most apparent, and the organ assumes more and more a uniform yellow tinge, from which the outlines of the lobules by degrees completely disappear. As regards the vascular apparatus of the liver, no important alterations are observed in the trunk and branches of the portal vein; they contain a small quantity of thin blood; in the hepatic veins I have found bundles and masses of tyrosine crystals, along with normal blood corpuscles. Attempts at injection have proved unsuccessful, the size which is injected into the hepatic and portal veins becoming extravasated, without penetrating into the capillaries, apparently because the delicate blood-vessels have lost their support, from the destruction of the secreting cells. In thin sections of the gland, the injection could be distinctly seen to fill only the capillaries nearest the central vein, and then to escape into the parenchyma. The centre of the lobules appeared of a dirty-yellow colour, and exhibited here and there brownish crystalline masses of leucine; at their periphery nothing but fine bluish-grey oil globules could be detected. Where the disease had run through its entire course, the hepatic cells were no longer visible; in place of them were found brown granules, and large particles of colouring-matter, oil globules, and isolated bodies similar to cell nuclei, often accompanied by needles of tyrosine and globules of leucine. It was only where the morbid process had been arrested at an early stage, that there could be seen isolated cells loaded with fat or pigment, and this was especially the case in the rounded margin of the right lobe. In the seventeen most recent observations of acute atrophy of the liver, the disintegration of the hepatic cells was always made out; in the earlier cases the organ was not examined with this object in view.

The gall-bladder was in most cases empty, containing only a small quantity of grey mucus, or a turbid, pale-yellow, rarely brown or greenish, fluid. The bile ducts exhibited nowhere any impediment to the excretion, and likewise contained no bile; their mucous membrane was in most cases of a grey colour, and their calibre seemed narrowed.

Along with the atrophy of the liver, the spleen, in most of the cases, was found considerably enlarged and congested; out of 23 cases in which this organ was carefully examined, in 19 it was enlarged, in 3 normal, and in 1 small. There are certain causes, such as thickening of the capsule, or profuse hæmorrhage from the roots of the portal vein, which may account for the non-appearance of the swelling. In some cases the mesenteric glands were also found to be enlarged.*

The stomach and intestinal canal present no important alteration of structure; the mucous membrane is here and there ecchymosed, but there are no losses of substance nor deposits in the solitary glands, or in Peyer's patches. The intestinal contents consist either of pale, dry fæces, or of black tarry matter, in which the microscope cannot detect any blood corpuscles which have not undergone some change.

The central organ and the larger trunks of the vascular system present nothing abnormal, with the exception of a jaundiced colour of the lining membrane, and a flabby, shrivelled character of the muscular tissue of the heart. The blood is of various characters; sometimes it is dark-violet and incompletely coagulated, but at other times hard firm coagula of fibrin separate from it; the number of colourless corpuscles is found to be increased in the blood in the right ventricle. The presence of large quantities of leucine and of urea, as in Observation XVI., is a much more remarkable circumstance.† The extravasations of blood in various organs and tissues are also remarkable. These were seen in the larger number of the cases; they were most frequent in the parts from which the portal vein draws its supplies of blood, from the surface of the mucous membrane of the stomach and bowels, more rarely in the tissue itself of these organs, beneath the serous coat of the intestine, and between the folds of the mesentery and omentum; they were also observed in the retro-peritoneal areolar tissue, and beneath the pleuræ and epicardium. The effusions into the serous cavities were often found tinged with blood. More rarely, hæmorrhages take place into the parenchyma of the internal organs, such as the lungs, kidneys, &c.

The kidneys have not received the attention which they deserve. Besides the deposit of pigment dependent upon the jaundice, I have found the glandular epithelium infiltrated with granules, and in most cases in a state of fatty degeneration, and the tissue itself flabby and shrivelled. The cases occurred for the most part among pregnant females, in whom Spaeth has made the same observation. Whether this abnormal condition is always present remains uncertain. The peculiar changes in the urine, the disappearance of the urea from it, and the accumulation of this substance in the blood, and further, the temporary occurrence of albuminuria, point, however, to an important implication of the kidneys.

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* Buhl; and Observation No. XVIII.

† Whether there were any other abnormal products present, such as the ammoniacal salts, has unfortunately not been investigated.

The central organs of the nervous system have in most cases presented abnormal conditions. In some cases the cerebral substance has appeared softened; a hydrocephalic softening has been particularly observed in the central parts (*Horaczek, Pleischl*); but it is still doubtful whether this condition is to be regarded, with Lebert, as the result of commencing putrefaction, or as a product of disease; at all events, this change in the consistence of the brain cannot account for the production of the nervous symptoms, as it is usually absent. The *post-mortem* examinations which I have myself performed have disclosed nothing abnormal in respect to the consistence of the brain, or to its amount of blood, even when death has been preceded by serious derangements of the nervous system.

VII.—*Nature of the Disease.*

We have now to construct a theory from the facts which have been observed; we must endeavour to explain the morbid process which takes place in the liver, and to point out the connection in which it stands to the concomitant symptoms.

Acute atrophy of the liver belongs to those obscure processes, as to the nature of which various opinions may be advanced, without its being possible for any one of them to obtain a general acknowledgment. The fact of the disappearance in a few days of one-half or one-third part of the original volume of a large gland abounding in blood, without any alteration in the blood-vessels leading to it, has a complete analogy in no other disease. Rokitansky, who was the first to give an accurate anatomical description of this affection of the liver, regarded the process as one of bilious liquefaction. An excess of the elements of bile, according to him, is formed in the blood of the portal vein, which, becoming separated, and pervading the entire vascular apparatus of the liver, causes destruction of the glandular substance by liquefaction. Such a mode of origin of the bile in the portal vein is opposed to our previous knowledge concerning the formation of this secretion, and, moreover, the increased secretion would not account for the destruction of the organ.

Henoch and Von Dusch likewise refer the destruction of the hepatic cells to the action of the bile. The former assumes the existence of a true polycholia, in consequence of which all the excretory ducts, even those of capillary fineness, become distended with secretion and compress the blood-vessels; hence arises a considerable impairment in the nutrition of the hepatic cells, which ultimately lead to their disintegration by fatty metamorphosis. Von Dusch is of opinion that the disease proceeds from paralysis of the bile ducts and lymphatic vessels, which gives rise to an infiltration of the organ with bile, and, through this, to a solution of the cells.

We cannot agree with either of these views, first, because no accumulation of bile can be observed to precede the atrophy; and, secondly, because, even if such were the case, it would not account for the atrophy. Henoch's assumption, that a state of polycholia induces the morbid process, does not harmonize with the symptoms of the preliminary stage, or, when this is wanting, with the commencement of the disease itself, inasmuch as the evacuations from the first contain but little bile; moreover, it cannot well be conceived, how an abundant formation of bile can distend the ducts as far as their capillary commencements, so long as there is no impediment to its excretion. The paralysis of the bile ducts and lymphatic vessels assumed to exist by Von Dusch, is purely hypothetical, and is not sufficient to account for a stoppage of bile, because the ducts at their commencement contain no muscular fibres, and the absorption of bile takes place more especially by the blood-vessels. But, even admitting that the atrophy was preceded by a stoppage of bile, this would afford no explanation of the rapid disappearance of the parenchyma of the liver, because, in cases where the ductus choledochus is obstructed, the hepatic ducts are not unfrequently filled and distended with bile during many months, and the hepatic cells are saturated with secretion, without any condition resembling acute atrophy resulting.* The experiments, therefore, by which Von Dusch has endeavoured to prove that bile exercises a solvent action over the hepatic cells, appear to be of doubtful value in framing a theory of the disease under consideration; moreover, on repeating these experiments, I have ascertained that the hepatic cells may be immersed in bile for whole days without undergoing solution.

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* See Observations V., VI., and VII.

Buhl regards the disease as analogous in its nature to typhus; the disintegration of the hepatic cells he attributes to the same cause as the concomitant hæmorrhages, namely, to the marked weakening of the heart's action, and to the rapid decrease of the peripheric metamorphosis of matter. The changes which the liver undergoes in typhus, pyæmia, &c., he considers as the commencement of acute atrophy.

In opposition to the theories just mentioned there is another, which attributes acute atrophy of the liver to a diffuse inflammation of the gland. This theory was first enunciated by Bright, who described cases of the disease under the appellation of hepatitis; in recent times, Engel, Wedl, and Bamberger have subscribed to it, and have explained the destruction of the cells by a fatty degeneration arising from an acute exudation-process.

Although I have some hesitation in identifying the destruction of the hepatic cells with fatty degeneration,—because in the case of other glands, such as the kidneys, fatty degeneration of the epithelium does not produce such a rapid and general destruction of the cells, and especially because, in acute atrophy of the liver, the fat can only be seen deposited in the circumference of the lobules, whilst the destruction of the cells is found to extend as far as the central vein,—still I cannot but adhere to this view in so far that, according to my experience, an exudation-process constitutes the starting-point of the disease. On close examination of a liver which is undergoing acute wasting, portions are found, mostly in the right lobe, in which the morbid process has not run its entire course. In such places, changes of structure may be detected, which appear to prove that the destruction of the glandular elements, and the collapse of the parenchyma, are preceded by hyperæmia and exudation. We here observe, not merely a remarkably congested condition of the capillaries, but, in the circumference of the lobules, broad grey bands, consisting of finely-granular matter with isolated cells undergoing disintegration, whilst the cells nearest to the central vein still continue normal, except that they are infiltrated with bile. At a later period the congestion disappears, the grey exudation matter is gradually removed, and the yellow debris of the secreting tissue becomes more closely aggregated; so that, in addition to the progressive atrophy of the organ, the outlines of the lobules are at last quite obliterated. The destruction of the cells as a result of exudation is partly owing to the fact that this is deposited in a close vascular network, where any effusion soon removes the necessary conditions of nutrition, and partly to the cell-walls being delicate, and the contents of the cells prone to decomposition.

The bile ducts at their origin are compressed at an early period by the exudation at the periphery of the lobules; as a consequence of this, the secretion formed in the central portion of the lobules stagnates, and passes into the central veins, and so into the general mass of the blood. It is in this way that jaundice comes to make its appearance, while the ochre-yellow colour of the liver and the pale mucous lining of the empty bile ducts owe their origin to the same cause. Against the supposition that an exudation process lies at the foundation of acute atrophy of the liver, it has been urged that no hyperæmic enlargement of the organ can be detected during the progress of the disease. This objection is of small weight, because the liver has rarely been closely examined previous to the appearance of the jaundice, and because no remarkable enlargement of the organ is necessary for the occurrence of diffuse exudation, and the less so, as this does not usually attack the entire gland at one time.

Another question which we must here endeavour to answer is:—how are the symptoms which accompany the disease connected with the structural changes found in the liver?

It has already been shown in what manner the jaundice arises; it is a more difficult matter to explain the mode of development of the nervous symptoms, the hæmorrhages and the tumefaction of the spleen.

I believe that the abnormal nervous symptoms, of which no explanation is furnished by any morbid appearance of either the brain or its membranes,* must be referred to changes in the composition of the blood. I do not refer here to the presence of the constituents of bile, as to the harmlessness of which I have convinced myself by a long series of injection-experiments; but I attribute the cause of the blood-intoxication to the complete arrest of the hepatic functions from the destruction of the secreting cells, and to the derangement of the renal secretion. The former of these causes includes not only the absorption of bile, and the retention in the blood of the substances intended for the formation of this secretion, but also the cessation of the powerful influence which the liver exerts over the processes of metamorphosis of matter, and the simultaneous passage of the disintegrated glandular substance into the blood. We are not acquainted in its entire extent with the influence exerted by the liver over the metamorphosis of matter; as yet we only know that the formation of sugar out of albuminous substances is a necessary link in the functional processes of the gland, and we infer from the existence of numerous other substances which have been observed, partly under normal, and partly under pathological conditions, such substances as xanthime, urea, inosite, leucine, tyrosine, cystine, &c., that the organ is intimately related in many ways to the metamorphosis of matter. The important nature of these relations is shown by the remarkable changes, which the urine—the general recipient of the chief ultimate products of this metamorphosis—undergoes in acute atrophy of the liver. The urea, which is the normal product of the disintegrated albuminous tissues, as we have seen, gradually disappears, and in its place, a large quantity of products which are foreign to healthy urine make their appearance. Its solid constituents consist almost exclusively of leucine and tryosine, together with a peculiar extractive matter; uric acid is present in tolerable quantity. It is doubtful what is the cause of the absence of urea. Is this substance really formed, although not excreted by the kidneys, or is the metamorphosis of tissue so far altered that at last no urea comes to be formed as an ultimate product? The considerable quantity of urea which is found in the blood, proves that its elimination is really stopped; still we must not conclude from this, that the formation of this product takes places in a normal manner, because we have no idea, not even an approximative one, as regards the amount to which it accumulates in the blood.*

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* Buhl is of opinion, that here, as in typhus, we can always detect an acute atrophy of the brain.

* It may be mentioned that in yellow fever also a considerable quantity of urea has been detected in the blood. If we assume that the formation of urea ceases, products should arise in its place, similar to those into which the albuminous substances are decomposed under the action of putrefaction, or upon the addition of nitric acid. The urea cannot be entirely replaced by leucine and tyrosine, because both these are poorer in nitrogen than albumen. It is thus clear, that along with these substances still another must be formed, containing a large proportion of nitrogen. Whether the amorphous matter which is found in large quantity in the

Thus far it must be regarded as an established fact, that acute atrophy of the liver induces very important abnormal conditions of the metamorphosis of matter, and that during its progress substances circulate in the blood which are not met with in that fluid in a healthy condition. What it is which induces the symptoms of blood-poisoning is uncertain; that it is not leucine or tyrosine is proved, by injections of these substances into the blood of animals producing no derangements of the nervous functions. It is more probable that they are due to a retention of the constituents of the urine; but this point cannot be determined without further investigation.

As regards the production of the splenic enlargement and the hæmorrhages, I believe that the former is to be accounted for, partly by the altered composition of the blood, and partly by the obstruction of the circulation in the hepatic capillaries, which lose their normal support in consequence of the disappearance of the cells; the circumstance of the hæmorrhages occurring chiefly in those parts from which the portal vein derives its blood, viz.: from the mucous membrane of the stomach and bowels, and between the folds of the omentum and of the mesentery, is in favour of a mechanical view of the matter. There must, however, be some farther reason to account for those hæmorrhages, which occur in other parts, such as the skin, &c. These have been attributed at one time to the debility of the heart's action, the general want of tone in the system, and the defective nutrition of the blood-vessels (*Buhl*), at another time, to the deficient formation of fibrine (*Monneret*), and at another, to an over-distended condition of the blood-vessels in consequence of a deficiency in the secretion of bile. There is another explanation which appears to me more probable, namely, an abnormal attraction between the walls of the vessels and the blood which has become altered in its composition, from which arise obstruction and rupture of the capillaries. Bernard* has already endeavoured to show, that the passage of sugar from the liver into the blood prevents the infiltration of the tissues, and promotes the circulation. In the cases before us, it is not only

urine corresponds to this, has not been ascertained with certainty; neither has any cause been assigned to prevent the farther transformation of this substance into urea. It is possible that the ferment matter, which experience shows to be contained in the liver, is indispensable to this further metamorphosis; but the subject can only be cleared up by new observations and direct experiments.

the formation of sugar which is arrested, but in addition to this, a whole series of substances pass into the blood, which may not be without some influence over the production of the hæmorrhages. It is obvious, however, that we do not possess a more complete proof of this theory, than of any of the others.

VIII. ETIOLOGY.

We are still without a clear insight into the mode of origin of acute atrophy of the liver; we can only enumerate in the meantime the circumstances under which the affection makes its appearance, without being able to state the respective share which these causes play in individual cases, or to trace more closely their mode of operation. Unfortunately, however, the same remark applies to the etiology of most diseases.

The disease is most frequently observed in the female sex; of the 31 cases, 9 were men and 22 females, so that the number of the latter more than doubled that of the former. Of the 22 females, one-half were attacked during pregnancy; consequently, more than one-third of all the cases were associated with this condition of the system, a circumstance which points to both of these conditions (sex and pregnancy) as predisposing causes. Nevertheless, acute atrophy of the liver is a rare affection, even in pregnant females; out of 33,000 cases, Spaeth found this complication present in only two instances. Very frequently during pregnancy, the kidneys and the liver become infiltrated with granular albuminous matter, which gives rise to alterations in the character of their secretions, and to fatty degeneration of the glandular epithelium. Under certain conditions this process advances to diffuse nephritis and hepatitis; it is generally known how frequently this happens in the case of the kidneys, and also, what are the symptoms which denote the presence of the renal affection; in the liver the process seldom attains such an advanced development, and here destruction of the gland is the result. Acute atrophy of the liver in pregnant females may be recognised by the fact that it is almost invariably accompanied by fatty degeneration of the kidneys. That it is not mechanical compression, as Scanzoni thinks, to which the cause of atrophy of the liver is to be attributed in pregnant females, is shown by the period at which this affection makes its appearance; in most cases, it is from the third to the sixth month, sometimes it is the seventh, but it is rarely at a time when the uterus can operate mechanically upon the liver.

As regards age, the period of life between 20 and 30 seems most predisposed to the disease. Of the 31 cases, there were:—

6	between 10	and 20	years	of age.
20	„ 20	„ 30	„	„
3	„ 30	„ 40	„	„
2	„ 40	„ 60	„	„

Cases of acute atrophy of the liver have also been described in children, but I know of no observation sufficiently detailed to find a place here.

Of the special injurious agencies, which precede the outbreak of the disease, and which after its appearance co-operate with its exciting causes, the following are the most conspicuous.

1. Mental emotions.—In several cases the disease appeared in individuals previously healthy, so immediately after a severe fright, or an outburst of passion, that the influence of mental emotions could scarcely be doubted. The patients became immediately jaundiced, began to be delirious and died in a few days. Such cases have been described by Vercelloni, Morgagni, Ballonius, and others.

2. Venereal excesses, syphilis, the improper use of mercury, drunkenness, and the other morbid influences arising from a dissolute life, have occasionally preceded the disease: still it is not certain that these morbid influences were connected with the disease as cause and effect, and it is still less possible to prove with certainty the influence of one of them apart from the others.

3. There would appear to be influences, of the nature of miasmata, confined to certain localities, which contribute to the production of the disease; at all events, we are led to believe in the existence of such influences from the presence of the malignant forms of jaundice, in different members of the same family, or in other individuals living in the same house, examples of which have been recorded by Budd, Griffin, and Hanlon. It still, however, remains a question, whether these cases ought, or ought not, to be regarded as examples of acute atrophy of the liver; sufficient anatomical evidence on the point does not exist. A miasmatic origin of the disease, as under certain circumstances seems at first-sight probable, may indicate rather the existence of one or other of the forms of bilious fever, which supposition is favoured by the frequency with which such cases have terminated favourably.

4. Typhus and the allied changes in the composition of the blood.—I have myself seen one case of acute atrophy of the liver proceeding from typhus, and Buhl has observed others, in which the mesenteric glands were enlarged in a similar manner to what they are in this disease. In such cases, there is a greater development of that infiltration of the hepatic parenchyma which we have already found to exist in typhoid jaundice. Usually no other cause can be detected, which can account for the origin of the morbid condition of the liver, and the etiology of the affection must be left for future investigations.

IX. DIAGNOSIS.

Acute atrophy of the liver is less easily recognised than it would appear to be. It is very frequently mistaken for other diseases, particularly typhus complicated with jaundice, bilious fevers of various sorts, pyæmia, &c., and such mistakes can only be avoided by a careful weighing of all the symptoms. During the preliminary stage, a diagnosis, as a general rule, is impossible. When hæmorrhages, violent headache, delirium, &c., are associated with jaundice, it then becomes a question whether, independently of the liver, there are local or general derangements present, which can or can not account for these symptoms. Typhus is recognised by the symptoms which mark its progress, the roseolar eruption, the bronchial catarrh, the diarrhœa, and the wandering character of the delirium; bilious fevers as a rule, are characterised by a more or less distinctly remittent type, and by repeated attacks of rigors; pyæmia is distinguished by rigors and by the presence of purulent deposits. Local diseases, such as meningitis, pneumonia, and peritonitis, which, when associated with jaundice and delirium, present a train of symptoms, resembling, as I have myself observed, those of acute atrophy of the liver, can usually be easily recognised by a careful examination of the individual organs. The characters exhibited by the liver itself are of greater importance in diagnosis, not so much the tenderness, which is now and then absent, as the diminution in volume, which advances rapidly, until at last the dulness on percussion completely disappears. The cautions which are to be observed in determining the dulness, have been explained in Chap. III. Of equal diagnostic value are the characters presented by the urine, the deposition of sediments of tyrosine, the crystalline forms which separate upon evaporation, &c. The importance of the remaining symptoms in diagnosis depends upon the frequency of their occurrence, a point which has already been discussed. Petechiæ and epistaxis, which in typhus are of frequent occurrence, appear to be of less importance than vomiting of blood, as indications of acute atrophy.

X. TREATMENT.

The results of treatment hitherto recorded, are, as has already been stated, of a hopeless nature; hence no approved empirical method exists. English physicians recommend emetics and purgatives, two classes of remedies, the energetic action of which upon the liver certainly cannot be denied. According to Corrigan, the progress of the disease is arrested by emetics, and according to Griffin and Hanlon, by drastic purgatives; in the case which I myself saw run a favourable course, purgatives and mineral acids were employed. This experience, small though it be, would be of great value if we could be sure that the observations really referred to cases of acute atrophy of the liver, which unfortunately is impossible, considering the uncertain nature of the diagnosis of the incipient stages of the disease. The lack of more direct experience must be supplied by general principles, and by the analogy of allied conditions.

In the preliminary stage, the same principles of treatment are of service, as in simple catarrhal jaundice; a more direct treatment is not called for, until symptoms denoting serious disease of the hepatic parenchyma manifest themselves. At the commencement, our object should be to remove the congestion and diffuse exudation; afterwards, when the atrophy has extended over most of the gland, no benefit can be expected from treatment. For the purpose just mentioned the stronger purgatives are especially recommended, by means of which the congestion of the liver is most effectually relieved, such for instance, as senna, aloes, colocynth, &c., and they should be administered in such doses as to bring about profuse evacuations. When there are severe pains in the liver, benefit will be derived from the application of leeches, cupping-glasses, and cold cloths, and in full-blooded individuals from venesection. When the symptoms of blood-poisoning and hæmorrhages make their appearance, the administration of the mineral acids is indicated, along with which purgatives may be continued, to keep up the action of the bowels. To check the vomiting we may prescribe ice, the magistery of bismuth,* and small doses of the aqueous extract of nux vomica. Hæmorrhages from the stomach and bowels require ice internally and externally, alum, gallic acid and similar astringents. When there are symptoms of nervous depression, we must employ stimulants, such as ether, camphor, and musk; in such cases, however, it is seldom that we can look for any good result.

In cases where the diagnosis is doubtful, especially where the distinction between acute atrophy and bilious fever remains uncertain, I would recommend large doses of quinine dissolved in acids.

Besides acute atrophy, there are other morbid processes, which, if they lead to complete disorganisation of the liver, and consequently to an arrest of its functions, may give rise to symptoms of blood-poisoning. Symptoms may appear under such circumstances which in most respects range themselves with those just described, but which differ in many points. Here, as in acute atrophy, there may be observed dangerous nervous symptoms, typhoid somnolence, delirium, coma, and convulsions, accompanied by petechiæ and ecchymoses of the skin, as well as by hæmorrhages from the mucous membrane of the stomach and bowels, and along with these symptoms we may find, in most cases, jaundice of a more or less intense degree; but on the other hand, the jaundice may be completely absent, the abnormal cerebral symptoms may be for the most part less violent, and, moreover, the characters presented by the preliminary stage may be very different from those of acute atrophy, as, for instance, when the disease is preceded for a long time by symptoms of occlusion of the bile ducts, or by those of cirrhosis or fatty degeneration of the organ.

Among the diseases of the liver, which may give rise to destruction of the glandular epithelium, may be mentioned, in the first place:—

B. 1. *Obstruction to the Flow of Bile resulting from Impermeability of the Ductus Choledochus and D. Hepaticus.*

In some cases, when this condition has lasted for several months, it gives rise to an atrophy of the gland, which in many points resembles acute atrophy. The organ diminishes in size and becomes soft and dry, the cells of the parenchyma which are infiltrated with bile become disintegrated into a finely-granular debris, mingled with oil globules and particles of pigment, while at the same time large quantities of leucine and tyrosine may be detected.

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* Trisnitrate.

Observation No. XIX.

Cancerous Deposit in the Duodenum.—Occlusion of the Ductus Choledochus.—Intense Jaundice.—Convulsions.—Coma.—Death.

Friedr. Bloch, a female, aged 58, who had been suffering for a long time from various diseases in Frankel's Asylum for Old People, was brought to the Jewish Hospital of this place on July 29th, 1853. The patient had her mental powers somewhat impaired, and was of a surly temper, so that it was impossible to ascertain any accurate history as to the mode of origin of her illness. On admission, she presented a series of symptoms, indicative of intense jaundice as a result of closure of the ductus choledochus.

The woman was emaciated, and her dry wrinkled skin was coloured at some places brownish-yellow, and at others olive-green; its temperature was low. The tongue was clean, and the appetite unimpaired. The bowels were confined, and the stools consisted of dry, clay-coloured scybala. The urine was brownish-black, and deposited at one time a brick-red, and, at other times, a yellow, sediment of uric acid. The pulse was 50 and weak. The left lobe of the liver extended about 5 centimètres (2 inches) beyond the margin of the false ribs, and reached upwards to a level with the lower margin of the sixth rib; the right lobe, which was elongated, and drawn downwards by means of a tight-lace constriction, extended 8 centimètres (3.15 inches) beyond the margin of the ribs. Close to the sternum, the hepatic dulness amounted to 9 centimètres (3.54 inches); in the mammary line it was 16 cent. (6.3 in.), and in the axillary line, 14 cent. (5.51 in.) The surface of the organ felt smooth; its margins were sharp towards the left side, but on the right side were somewhat rounded. Ten centimètres (4 inches) to the right of the umbilicus, and somewhat deeper, there lay a soft, pear-shaped tumour, the size of a duck's-egg, which was evidently the distended gall-bladder. No tumour capable of compressing the ductus choledochus could be made out upon palpation.* No hardness could be felt in the region of the pylorus and of the head of the pancreas. There never had been any symptoms of colic from gall-stones.

The patient was treated for a long time with tincture of colocynth, aloes, and such-like remedies, along with warm-baths and easily-digested food, without any marked change in her condition. From time to time she suffered from severe cardialgia, which yielded to the use of the magistery of bismuth (trisnitrate) and belladonna.

The emaciation gradually increased, the feet became œdematous, the quantity of brownish-black urine was reduced, but remained free from albumen; the mental powers at the same time became more and more depressed. The patient continued in a state of gloomy silence, answered questions either not at all or incompletely, asked for nothing, complained of nothing, and ate the nourishment offered her with a slight appetite. She was generally asleep; on calling loudly to her, she awoke, opened her dull, lustreless eyes, said she felt well, and immediately went to sleep again.

Towards the end of October, she had repeated attacks of epistaxis, which were arrested with difficulty.

At the end of November, she was suddenly seized with convulsions; the attacks lasted from a quarter to half-an-hour, and returned from time to time. The somnolence now passed into deep coma, from which she could no longer be roused.

The patient now became rapidly collapsed; her skin became shrivelled like that of a mummy, and its temperature sank more and more; the pulse grew smaller, and at last was imperceptible, until death took place on the 23rd of December.

Unfortunately only a very imperfect *post-mortem* examination could be made. The liver, stomach, duodenum, and pancreas were the only parts carefully examined.

The liver was somewhat enlarged, and its right lobe was subdivided by a tight-lace fissure. Its surface was smooth; its margins were sharp, except those of the right tight-lace lobe, which were rounded. The gall-bladder projected 2½ inches beyond the margin of the liver, and contained about 9 ounces of turbid dark-brown bile. The gall ducts, from the ductus choledochus and ductus hepaticus, as far as their finest terminal branches, contained a quantity of turbid brown, thin fluid, of which from 18 to 20 ounces could be collected on cutting up the organ. The ducts were all considerably enlarged. The hepatic duct measured 1 inch and 4 lines, and was marked by numerous deep pouch-like depressions, leading into the adjoining branches. The parenchyma of the glands was of a dark-brown colour, soft and easily compressible; and the outlines of the lobules were indistinct. Upon the cut surface were seen small greyish-white, hard deposits, partly rounded and partly branched, like a tree, which corresponded with the course of the finer twigs of the hepatic vein, and in some places completely filled these twigs. Crystalline deposits of the same substance lay embedded in the walls of the larger branches of the hepatic veins, so firmly, that on scraping with a knife they could only be separated with difficulty. These crystalline masses were made up of an aggregation of fine needles. No abnormal deposits could be detected in the ramifications of the portal vein or of the hepatic artery. The cells of the hepatic parenchyma were in a great measure disintegrated; on microscopic examination only a few were found to remain intact: in their place were seen fine brown molecules and oil globules, and also numerous bundles of acicular crystals (tyrosine), and globular masses deposited in concentric layers (leucine).† These deposits were at some places densely aggregated, but at other parts were either very scanty or altogether absent.

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* Firm pressure upon the abdominal walls was made with difficulty, on account of the obstinacy with which the woman kept her muscles in a state of constant contraction.

The same crystalline and concretion-like deposits, as were found in the liver, were also present in the turbid bile, along with tables of cholesterine. A large quantity of leucine and tyrosine was obtained from the substance of the liver, the identity of which was proved by Professor Staedeler, on elementary analysis.* In the mother-liquid also, from which these bodies had been separated, a substance was observed, which, upon exposure to the air, assumed an intense blue colour, similar to Chromogen, with which we shall afterwards become acquainted, when considering the transformation of the biliary acid into pigment. The liver did not contain any sugar.

At the point of entrance of the ductus choledochus into the duodenum there was a tumour the size of a walnut, with all the characters of villous cancer. By means of this the bile duct was obliterated. The pancreas was flabby and shrivelled; the mucous membrane of the stomach, at some places, was ecchymosed, but without any loss of substance. The spleen was somewhat enlarged and anæmic.

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* See Müller's Archiv f. Anat., &c., 1854. † See Frontispiece, Fig. 4.

Large quantities of the brownish-black urine passed by the patient were examined for the presence of the biliary acids, but always with a negative result. It was not examined for leucine and tyrosine.

The morbid process just described is, on the whole, rare; in most of the cases where death has followed as a consequence of occlusion of the bile ducts, the hepatic cells, on *post-mortem* examination, were infiltrated with colouring-matter, but were otherwise unaltered. The manner in which they become disintegrated is still unknown; this disintegration cannot be attributed with certainty either to the solvent action of the bile, or to the destruction of the branches of the portal vein by the enlarged bile ducts, inasmuch as we often observe the same infiltration of the parenchyma, and enormous enlargement of the ducts, without any such consequences.

Whether the derangements of the nervous functions always present the same characters as in the case just communicated must be determined by future experience; this may depend upon the more or less complete disintegration of the hepatic cells, and upon the rapidity with which it takes place. Budd has described a case in which pain in the liver, hæmatemesis, headache, and other symptoms made their appearance, but where delirium was only present in the night-time during the last week of life.*

c. *Acholia from Cirrhosis.*

Cases have repeatedly occurred to me, in which individuals who for a long period have suffered from cirrhosis of the liver, have suddenly presented a series of morbid symptoms which are foreign to that disease. They have become unconscious, and have been afterwards seized with noisy delirium, from which they passed to deep coma, and in this state have died. In one case there were spasmodic contractions of the muscles of the left side of the face. In most cases, slight jaundice made its appearance at the same time, and in one instance there were petechiæ. Upon *post-mortem* examination, not the slightest lesion could be detected in the brain; neither were there indications of any acute disease which could account for the derangement of the cerebral functions. The liver, in all the cases, presented cirrhotic degeneration in a marked degree, and the glandular cells were for the most part loaded with fat; large quantities of leucine separated from it; the bile ducts contained only a small quantity of pale bile.

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* Budd draws the conclusion from this observation, that the secreting functions of the liver are not indispensable to the continuance of life, and that a patient may live for a lengthened period after the destruction of the hepatic cells; but we are not entitled to make such an assumption.

OBSERVATION No. XX.

Ascites.—Anasarca.—Diarrhœa.—Delirium.—Coma.

Cirrhosis of Liver.—Deposits of Leucine in the hepatic veins.—The central organs of the nervous system normal.

E. Radesey, a bookbinder, aged 59, came to the Hospital on the 4th of December, 1854, with great œdema of the feet, and ascites. The heart and lungs were healthy; the hepatic dulness could not be distinguished; the size of the spleen could not be determined, owing to its situation being abnormal; the veins on the right side of the abdomen were greatly enlarged. The appetite was unimpaired; the stools were thin and pale; the urine scanty, red, and without albumen. The patient confessed that formerly he had been in the habit of drinking much brandy, and could account for his illness in no other way. He was ordered decoction of colocynth. Copious thin evacuations diminished the size of the abdomen, and the patient felt himself better. On the 17th, he suddenly fell into a state of unconsciousness, from which he could not be roused; the countenance became pale and sunk; the pupils were of normal size, and moveable; the pulse was 70, and full; stools involuntary; no vomiting. Was ordered infusion of Arnica flowers, with spirit of nitric ether.

On the 18th, great restlessness; inarticulate cries; complete loss of consciousness; pulse 90; respirations 22. About noon, the mouth was observed to be drawn towards the right, as in paralysis of the facial nerve.

On the 19th, pulse 120; deep coma, and stertorous respiration.

On the 20th, tracheal râles, and death about noon.

Autopsy.

The membranes and substance of the brain were perfectly normal;

the lungs, moderately œdematous. Liver small; its outer surface, as well as that seen upon section, covered with cirrhotic nodules, separated by grey areolar tissue; the hepatic cells loaded with oil, and in some places with pigment. Granules of a sulphur-yellow colour, and with a diameter measuring up to a quarter of a line, were seen upon the inner surface of the hepatic veins. These were firmly adherent to the wall of the vessel, and were densely aggregated, so as to form a pellicle like that of hoar-frost. They were made up of a collection of globular masses of leucine, along with which were observed paler granules and needles, which were insoluble in alcohol, but were easily dissolved in ammonia. The gall-bladder contained a small quantity of orange-yellow fluid.

The peritoneum and serous covering of the bowels were covered with black specks. The mucous membrane of the stomach and intestine was pale and œdematous, the mesenteric glands were small; the kidneys and urinary passages were normal.

Observation No. XXI.

Ascites.—Diarrhœa.—Unconsciousness.—Coma.

Cirrhosis of the Liver.—Leucine in the Blood and Urine.—Brain normal.

Dav. Kliesch, a watchman, aged 53, stated that he had always led a temperate life, and that up to 1855 he had enjoyed good health. In November, he was seized with diarrhœa, which proved obstinate, and was accompanied by ascites, but not by œdema of the feet. The appetite remained unimpaired, but the vital powers gradually sank. The heart was normal; pulse 90; slight bronchial catarrh. Hepatic dulness diminished; spleen considerably enlarged. There were from four to six thin stools in the day, containing little bile; urine pale and containing no albumen; skin pale; abdominal veins not distended. Was ordered decoction of calumba root with tincture of nux vomica. The appetite continued moderate, and the patient's condition underwent little change, except that the ascites increased.

On February 4th, he became unconscious; the features were sunk; but the pupils remained unchanged. Pulse 86.

On the 5th, complete coma; a faint jaundiced tinge of the skin; several petechiæ. Pulse 120.

On the 6th, involuntary evacuations; pulse 130; tracheal râles. Death on the morning of the 8th.

Autopsy.

The membranes of the brain were moderately congested; the substance of the brain was unaltered; some of the blood-vessels were atheromatous. The lungs were congested and œdematous. In the heart there was a little loosely coagulated blood, in which could be detected 1.10 per cent. of fat containing cholesterine, and a large quantity of leucine. The lining membrane of the stomach was puffy, and of a livid colour. The mucous membrane of the upper portion of the intestine presented the appearance of recent congestion, that of the lower was of a slaty-grey colour; in the sigmoid flexure there were a few superficial ulcers, the size of a linseed. The fæces were thin and pale.

The spleen was enlarged by one-half; its capsule was thick, and its parenchyma of a uniform dark-brown colour. The liver was small; and its capsule opaque and thickened. Its outer surface, as well as that seen on section, was covered with little nodules; the organ was tenacious (*zähe),* but flabby and shrivelled. The gall-bladder contained a small quantity of thin pale bile. The hepatic cells contained a large quantity of oil. The uro-poietic system was normal.

The urine obtained from the dead body had a specific gravity of 1011; it was acid, and presented the reaction of bile-pigment in a slight degree. It contained traces of albumen, and along with urea, a moderate quantity of leucine. This last was found in larger quantity in the parenchyma of the liver.

D. *Acholia from Fatty Degeneration of Liver.*

Only in one case of this form of degeneration have cerebral symptoms been observed. This was a female, in whom the accumulation of fat in the liver became so great, that the secretion of the gland was reduced to a minimum.

Observation No. XXII.

Jaundice of fourteen days' duration.—Somnolence.—Vomiting.—Sudden supervention of violent delirium.—Coma.—Death.

Fatty degeneration of the Liver in its most advanced form.—Enlargement of the Spleen.

Louise Fischer, a washerwoman, 44 years of age, was admitted in a jaundiced condition, on the 20th of May, 1856. The jaundice had commenced 14 days before, and for 8 days the patient had been obliged to keep her bed. During this time, she had been feverish, and had suffered from profuse mucous diarrhœa; the urinary secretion was scanty. Upon examination, we found the temperature elevated, the pulse 128 and small, no abnormal sound of the heart, and the respiratory organs healthy; the abdomen was distended from tympanites; the liver reached as high as the lower margin of the fifth rib, and from this point, in a line with the mamma, extended 17 centimètres (6.69 Eng. inches) downwards; the spleen was likewise considerably enlarged. The stools were frequent, thin, and of a greyish-yellow colour; mucous vomiting occurred three times in the course of the day. The urine, which had to be drawn off by catheter, was feebly acid, and assumed a greenish colour upon the addition of nitric acid; neither albumen nor the bile acids could be detected in it; but upon further examination of it, globular masses similar to hypoxanthine, and traces of leucine, were discovered. She was ordered muriatic acid in decoction of mallows.

Towards evening, the patient, who, even at the time of her admission, was somewhat unconscious, was suddenly seized with violen delirium; she raged and shouted during the whole night; on the following morning collapse set in; the pulse became thready, and the extremities cool.

Death took place at 10 A.M. of the 21st.

Autopsy, seven hours after death.

The membranes of the brain were yellow; the sinuses contained firmly-coagulated blood; the substance of the brain was moderately congested, and of normal consistence. Both lungs were free from adhesions, congested, and slightly œdematous, posteriorly and inferiorly.

There was a small quantity of blood in the heart, some of it loosely, and the rest firmly coagulated.

The mucous membrane of the stomach and intestines was pale; its glands were not enlarged; there was some thin grey fæcal matter in the ileum and colon; the mucous membrane of the sigmoid flexure was recently injected and puffy.

The spleen was large; weighed 0.372 kilogr. (13 oz. avoird.): it was soft and of a reddish-brown colour.

The liver was considerably enlarged; weighed 3.23 kilogr. (7 lbs. 2 oz. avoird.); its margins were sharp, and its colour waxy-yellow. Its cut surface appeared remarkably anæmic; the circumference of the lobules was pale-yellow, their centre was greenish-yellow; the hepatic cells in the latter situation were loaded with bile-pigment, whilst in the former, they were distended with oil. The portal vein was unobstructed, and the branches of this vessel, as well as those of the hepatic veins, could be readily filled with coloured injection. The gall-bladder contained a small quantity of clear, pale, mucous fluid. The bile ducts were normal, and no obstruction could be detected either in them or in Glisson's capsule, or in the duodenum. The substance of the liver consisted in great part of fat; it contained 78.07 per cent. of fat, and 21.93 p. c. of areolar tissue; along with the fat, were found large quantities of leucine. In the solid residuum of the blood obtained from the right side of the heart, were found 1.91 per cent. of fat, and an amorphous yellow-green colouring-matter, together with traces of leucine.

Other forms of disease might be included with those just described, which terminate in a similar manner, by suspension of the functions of the liver. Thus I have had under my care a lady, aged 53, with a very extensive cancer of the liver, as shown by palpation, who was suddenly seized with delirium, convulsions, and coma; whilst at the same time the skin became slightly jaundiced, and covered with petechiæ. The stools, which at first were pale, and deficient in bile, assumed a brownish-black colour, in consequence of hæmorrhage from the bowels. Death took place in the midst of profuse epistaxis. Unfortunately no *post-mortem* examination could be made in this case.

Acholia must always be expected, when the structure of the liver has undergone such extensive changes from acute or chronic diseases, that the function of the organ is necessarily completely arrested.

CHAPTER VI.

CHRONIC ATROPHY OF THE LIVER.

1. *Causes and Mode of Production.*

THE size of the liver in health, is subject to considerable variations, the limits of which we attempted to define in the second chapter. Independently of this, there are numerous pathological derangements of its nutrition which reduce the size of the gland, and give rise to a corresponding diminution of its functional value. These may result from anything which permanently impairs or arrests the circulation through the capillary system of the gland.* Hence we observe general or partial wasting of the liver in the course of various structural diseases of the organ, as one of the phenomena, and at the same time as a necessary result of the primary disease;

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* The liver, like the lungs, possesses a double vascular apparatus, a nutritive and a functional; the hepatic artery serves the former purpose, the vena porta the latter. Hence impediments to the nutrition of the gland, so far as these are dependent upon the flow of blood, should proceed from the hepatic artery; this, however, is contrary to experience. A rigid separation of the two functions does not exist in nature; but not, apparently, because the capillaries of the two vascular systems anastomose. Obliteration of the hepatic artery is not followed by arrest of nutrition (LEDIEU, *Journ. de Méd. de Bordeaux, Mars,* 1856; GINTRAC, *L'oblitération de la veine porte,* Bord., 1856, p. 51). Occlusion of the portal vein is not followed by a cessation of the secretion of the liver (GINTRAC; *also my own Observations,* Nos. XXIX and XXX). So far as our present experience extends, the portal vein exercises a more powerful influence over the preservation of the normal volume of the liver than the hepatic artery, which, for the most part, supplies only the walls of the bile ducts and blood-vessels, and penetrates but a short distance into the interior of the lobules. Diseases of the hepatic artery and its branches, of such a nature as to obstruct the flow of blood, are but little known, and, indeed, have been but little sought for. I have myself frequently observed only one alteration in these vessels as of frequent occurrence, viz., an accumulation of black pigment. We have no experience as to the influence of functional derangements of the hepatic plexus of nerves, over the nutrition of the parenchyma of the liver.

not unfrequently, however, the wasting is independent of such a cause. We do not at all refer here to those forms of atrophy which result from the development of new growths in the liver, such as echinococci or cancerous deposits, or which are dependent upon enlargement of the bile ducts, or upon cirrhotic wasting and induration; neither do we refer to the partial atrophy produced by the cicatrisation of abscesses, or by the obliteration of the larger branches of the portal vein, or lastly, to the atrophy of the liver which follows the enlargement of the hepatic veins in mechanical hyperæmia: all these forms can be afterwards discussed more in detail under the head of the respective diseases which give rise to them. We confine ourselves at present to those forms of atrophy which have an independent existence, are accompanied by no other important change of structure, and hence lay claim to a certain degree of individuality. This form of atrophy makes its appearance under very various conditions.

The first cause which we recognise is the compression of the organ from without, from which results an amount of atrophy corresponding to the extent and force of the pressure. Bearing upon this there are the familiar effects of tight-lace chest, already mentioned under the head of 'Diagnosis'. Besides the alteration in the situation of the liver, tight-lacing produces more or less deep depressions on its surface; and the glandular parenchyma at the parts corresponding to these depressions dwindles into a mere bridge-like process penetrated by the enlarged vessels and bile ducts; at the same time, the organ in many cases becomes shrivelled and collapsed. The loss of substance which the gland undergoes from this cause is, as a general rule, but very slight; it is a removal (*Verdrängung*) of the parenchyma, which takes place, rather than an atrophy.

Occasionally the liver suffers more from compression of its convex surface by extensive pleuritic or pericardial effusions, especially when

FIG. 24.

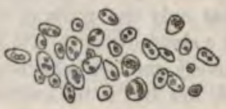

FIG. 24. Secreting cells of a liver, which had undergone chronic atrophy. The cells were much reduced in size, measuring only from $\frac{1}{230}$ to $\frac{1}{130}$ of a Paris line. They have also lost their normal granular contents.

the gland is firmly united to the diaphragm by short adhesions. Under such circumstances, extensive depressions are not unfrequently produced on the surface of the gland; the parenchyma at the places corresponding to these depressions assumes a dark-brown colour; the cells become smaller and lose their granular contents; whilst at the same time a few brown granules make their appearance in their interior. (Fig. 24.)

A similar result is produced by circumscribed peritoneal exudations, which occasionally leave behind deep depressions on the convex surface of the liver. (Fig. 25.)

FIG. 25.

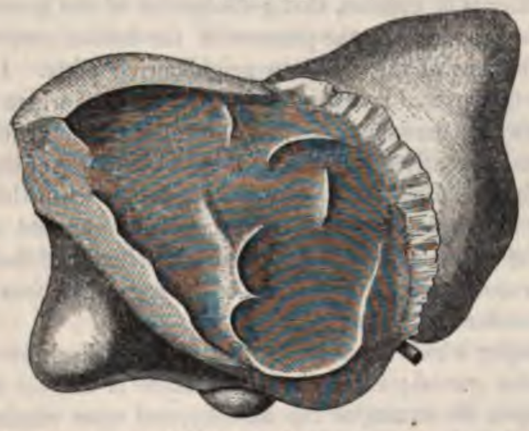

FIG. 25. Chronic atrophy of the liver, resulting from the external compression of peritoneal exudations, circumscribed between the concave surface of the diaphragm and the upper surface of the gland.

Cruveilhier* observed a considerable depression resulting from the compression of an hypertrophied heart, the gland being at the same time firmly adherent to the diaphragm.

Similar consequences may arise from enlargement of those portions of the intestine which are nearest to the liver, if, from their being distended with gas or fæcal matter, they exert a permanent pressure upon the organ. In this way, I have seen an extensive diminution of the liver produced by an enormous enlargement of the curve of the colon. The case was that of a man, aged 36, in whom a circumscribed peritoneal exudation had formed in consequence of a chronic perforating ulcer of the stomach. This gave rise to a constriction of the left curve of the colon, and to a stagnation of the gaseous and solid contents of the transverse and ascending colon. By this enlargement of the bowel the liver was pushed towards the ribs, high up into the right hollow of the diaphragm; the left lobe, which was situated nearest to the compressing cause, as also a portion of the right lobe, were atrophied. Budd[*] describes a partial atrophy of the gland in a preparation preserved in the Museum of King's College, which was obtained from a patient labouring under paraplegia, in whom the large intestines had for a long period been distended.

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* Anat. pathol. génér., Tom. III., p. 208.

Cruveilhier is of opinion, that a diminution of the parenchyma of the liver is produced by the pressure of the fluid of ascites, as also by the adhesions of the organ to neighbouring parts. I have not been able to convince myself of this, even in cases where there has been the most extensive ascites and the most numerous adhesions. I have not unfrequently found the organ small, but often of normal size, or even enlarged. In chronic peritonitis, it is only when the liver has for a long time been covered with a quantity of purulent exudation, or when the inflammation has extended to the fissure of the liver and Glisson's capsule, that a diminution of the organ has been observed to be of constant occurrence.

The atrophy which arises in the way just described, as a general rule, remains partial; its clinical importance is usually small, and depends upon the extent of the atrophy, and upon whether or not the larger bile ducts and blood-vessels have been subjected to the pressure.

The diminution of the liver, which extends throughout the entire organ, is of much more importance than this atrophy from compression, both as regards its consequences upon the entire organism and its local effects on the portal system. The liver here diminishes in every direction; its weight sinks to one-half the normal weight, or even to less.[†] The surface of the gland is smooth or slightly granular, or marked by linear wrinkles, and in some cases presents likewise distinct, isolated depressions; the parenchyma is dark reddish-brown, and is sometimes spotted with greenish-brown or yellow deposits of fat. In most cases, no trace of the lobules can be distinguished in the uniformly brown surface; when they are observed, they appear smaller than in the healthy gland.*

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[*] "On Diseases of Liver."

[†] In one female, aged 26, the weight was 0.7 kilogr. (1 lb. 8½ oz. avoird.); in a man, 53 years of age, it was 0.85 kilog. (1 lb. 14 oz. avoird.); in a woman of 50, it was 0.86 kilogr. (1 lb. 14⅓ oz. av.); and in a man aged 59, it was 0.62 kg. (1 lb. 6 oz. avoird.).

Important alterations may be distinguished in the blood-vessels: the portal vein is usually considerably enlarged as far as its subdivision into capillaries at the periphery of the lobules, at which place the enlargement terminates by a club-shaped extremity. The walls of the enlarged veins are sometimes normal, but at other times they present a remarkable thickening of the sheath, formed by Glisson's capsule.† This thickening disappears suddenly where the capillary ramifications commence. The capillaries themselves are in a great measure destroyed; they become filled with brown molecules, or they sometimes contain flakes or granules of black pigment; hence injections of the portal vein usually succeed very imperfectly; it is only here and there that a few isolated capillaries become filled as far as their anastomosis with the roots of the hepatic veins; the latter are usually more easily injected, and the injection runs to a greater distance. The meshes formed by the capillary vessels and by the enveloping areolar tissue are contracted, and at some places completely disappear, so that the walls of the obsolete vessels are in immediate contact with one another. Small atrophied cells are here and there observed in the narrow meshes.

The hepatic veins in many cases participate in the enlargement of the vena porta, but always in a less degree; their walls for the most part continue thin, and hence their bluish colour forms a marked contrast with the yellowish-red of the walls of the portal vein. The hepatic artery in two cases has appeared somewhat smaller than natural; Glisson's capsule has frequently been found thickened. The hepatic cells, which at some parts of the organ completely disappear, are distinctly visible at other parts, but are always pale, without any granular contents, with puckered walls and angular outlines; for the most part they are very small; they often contain brown pigment granules, which occasionally fill up completely the cavities of the cells, or little rods of the brown colouring-matter of bile. In many cases* cells are found filled with oil, sometimes scattered throughout the entire organ, at other times in isolated deposits. In the bile ducts there is only a small quantity of a pale secretion, which frequently contains albumen.

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* The thickness of the parenchyma between a central and an interlobular vein measures from ⅓ to ¾ of a millimètre; whilst in other cases this space is 1, 1½, or 2 millimètres in breadth.

† In 6 out of 18 cases.

Together with these changes in the liver, I have repeatedly observed enlargements of the veins of the stomach and large intestine, as also subserous ecchymoses and mechanical congestions of the spleen. This last was present in seven out of eighteen cases. Ulcerative processes in the stomach and intestine were present in 8 cases, 3 of which were caused by chronic dysentery; 2, by simple chronic ulcer of the stomach; and 3, by cancerous ulceration; in one case cord-like thickenings in the mesentery, with obliteration of isolated venous branches, could be detected. As consecutive changes, ascites and anasarca were found twelve times, and acute peritonitis twice.

Observation No. XXIII.

Chronic Atrophy of the Liver, with considerable enlargement of the branches of the portal vein.—A small ulcer at the pylorus, without any constriction of this opening. Distinctly visible, peristaltic movements of the Stomach.—Death from exhaustion.

Adam Blaschefsky, a day-labourer, aged 53, was admitted on the 21st of November, 1854.

The patient was emaciated, but free from œdema and from any abnormal colouring of the skin; he had complained for a long time of pains in the epigastrium, and had often brought up the food which he swallowed, without the admixture of any foreign substances. His appetite was but slightly impaired; the stools were regular, of normal consistence, but pale. The thoracic organs were intact, the respiration free, the heart's action normal; pulse 62. The abdomen was collapsed, and the abdominal walls when felt, appeared unusually thin. Upon closer examination, the contour of the stomach distended with gas was distinctly visible; on percussion, the full tympanitic sound of this organ could be distinguished from the more abrupt sound of the adjoining small intestine. Half-an-inch to the right and above the umbilicus, was observed a somewhat prominent tumour, which felt hard and uneven, was tender upon pressure, and could be shifted about. This was of course assumed to be a cancer of the pylorus.

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* In 5 out of 15 cases.

The hepatic dulness was reduced; in the median line it amounted to 2, in the mammary line to 3, and in the axillary line to 5 centimètres. ($\frac{4}{5}$, $1\frac{1}{5}$, and 2 inches.)

Under the use of tincture of rhubarb and extract of belladonna, the vomiting subsided; but the emaciation increased with tolerable rapidity, notwithstanding sufficient nourishment and an improved digestion. The tumour at the pylorus changed its position repeatedly; it was found sometimes to the right, and sometimes to the left, of the umbilicus, and at other times beneath it, right in front of the vertebral column. During the process of digestion, the peristaltic movement of the stomach could be observed most distinctly. A swelling first formed at the margin of the left ribs, corresponding to the great cul-de-sac of the stomach, which moved slowly towards the pylorus, and from this returned towards the left side. Behind the swelling, there was observed an annular constriction, by means of which it seemed to be pushed forwards. Not unfrequently, a second swelling followed the first, and was separated from it by a constriction; in such cases, the stomach appeared divided into two halves. Each of these movements of the stomach lasted from one-half, to an entire, minute, and between them there were intervals of four, or sometimes six minutes. Occasionally the bulging passed beyond the pylorus, which then became more elevated, apparently from the passage of the contents of the stomach into the duodenum. No movement could ever be perceived in the empty stomach. So long as there was any chyme present, the contractions could be produced by giving Hungarian wine, by percussion, and by repeated palpation of the stomach; they always presented the same characters, except that the interval between two movements was somewhat shorter when they were excited by the causes just mentioned.

On the 22nd of December the patient, who felt comparatively well, asked for his discharge; but so soon as the 27th he returned in a state of great exhaustion. Excesses in diet during the Christmas festivities had induced severe catarrh of the stomach; the tongue was thickly coated; there was great tenderness of the epigastrium, and no appetite. Vomiting of mucous matter, without any admixture of blood, suddenly supervened; the bowels were confined. Mild neutral salts and bitter, slightly astringent medicines, along with a suitable diet, failed in checking the catarrh; the patient became rapidly collapsed, and died from exhaustion on the 14th of January.

Autopsy on January 15th, 1855.

The body was greatly emaciated, and weighed 45 kilogrammes (99 lbs. avoir.)

The brain and its membranes were anæmic, but in other respects normal. The mucous membrane of the bronchi was pale; the lungs were collapsed and anæmic, except in the dependent portions, where there was a small amount of hypostatic œdema. The heart was small and covered with opaque fibrous spots (*Sehnenflecken*); its valvular apparatus was normal. The mucous membrane of the pharynx and œsophagus was pale; the abdominal walls were unusually thin. In consequence of the *post-mortem* rigidity, the stomach lay closely contracted beneath the liver, and did not extend so far downwards as during life. Immediately beneath the stomach lay the transverse colon, which was tightly contracted; the small intestines had completely fallen down into the abdominal cavity, so that the abdominal walls were in contact with the spine, and only separated from it by the thin omentum and mesentery. It was owing to this abnormal distribution of the small intestine, and to the unusual laxness of the abominal muscles, that we had been able to observe the movements of the stomach so distinctly during life.

The spleen was firmly adherent to the hollow of the diaphragm; its capsule was covered with an extensive white membrane, 1½ line in thickness; the parenchyma was anæmic.

The lining membrane of the stomach was tumid, of a dark, livid hue, and covered with a layer of mucus. The veins, especially those of the small curvature, were much enlarged and distended, and dark-blue. Just before coming to the pylorus, there was an ulcer half-an-inch long, 3 lines deep, and equally broad, the margins of which were still covered with puckered mucous membrane, and which presented a slightly-elevated edge, formed of cancerous infiltration of the submucous areolar tissue. The muscular tissue throughout the entire stomach, but especially at the pyloric opening, was hypertrophied.

The mucous membrane of the small and large intestines was somewhat darker than usual, and its veins were enlarged; the fæces were solid and of a yellow colour.

The uro-poietic system presented nothing abnormal.

The liver exhibited all the characters of chronic atrophy in a marked degree. It was small,* flabby, and tenacious *(zähe)*; its outer surface was uneven, and covered with flattened prominences about quarter of a line in breadth. Its cut surface discharged an enormous quantity of thin fluid blood from numerous widely-gaping mouths of vessels, which were everywhere visible. The calibre of these vessels was considerably enlarged. The left branch of the portal vein measured nearly 1½ Paris inch, and one of its branches, 8 lines. This enlargement extended throughout the entire subdivisions of the portal vein as far as its capillary ramifications, and could also be observed in the hepatic veins, although to a less extent. The tunica adventitia of the portal veins was very considerably thickened; its ramifications in the substance of the liver were of a reddish-yellow colour, and consequently presented a striking contrast to the thinner bluish-white branches of the hepatic veins. In tracing the ramifications of the portal vein, several of its branches (and at the same time apparently also branches of the hepatic artery and of the hepatic duct,) were found enclosed in a thick common sheath of areolar tissue, which likewise contained a network of elastic fibres, each individual vessel being furnished, in addition, with a tunica adventitia of its own. Interspersed here and there through these sheaths could be seen portions of substance exactly corresponding to the hepatic tissue, which were observed lying between the ramifications of the portal vein. Hence the approximation of several branches of the portal vein appeared to depend upon the disappearance of the intervening portions of hepatic tissue. The thickening of the sheaths of the vessels extended as far as their finer ramifications, and only terminated when these became lost in capillaries. In thin sections (Fig. 26) of the dried hepatic tissue, thick sheaths could be seen everywhere surrounding the apertures of the vessels, some of which were round, and others elongated; these sheaths occasionally enclosed branches of the hepatic artery, and contrasted remarkably with the thin walled branches of the hepatic vein.

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* The weight of the liver amounted to 0.85 kilogr. (1 lb. 14 oz. avoird.), the weight of the body being 45 kilogr. (99 lbs. av.), thus making the relative weight of the two as 1 to 50.3. The weight of the spleen was 0.18 kilogr. (6.35 oz. av.), and its comparative weight with that of the liver, 1 to 4.7. The left lobe of the liver measured transversely 3¼ inches, and from before backwarks 3¼; the right lobe measured 5½ inches transversely, and 6 from before backwards; the maximum thickness of the organ amounted to 2 inches.

The hepatic parenchyma was of a dark reddish-brown colour, and nowhere exhibited a distinct subdivision into lobules. When fine sections of it were submitted to microscopic examination, they exhibited a tolerably distinct and regular network, and also at some places ramifying twigs, where these happened to run in the same plane with one another over a tolerably extensive space. This network was made up of a connected system of small tubes containing small yellow, or yellowish-brown, or occasionally reddish-brown granules. In the spaces included in the meshes of this network (some of which were elongated, others oval, and others rounded), except where the tubes were closely compressed, small pale cells were observed, some of them containing nuclei; these cells, after repeated washing, could be removed from the fine sections. At first it appeared doubtful in what light this network was to be regarded, whether it was formed by the ramifications of the blood-vessels, or by the

FIG. 26.

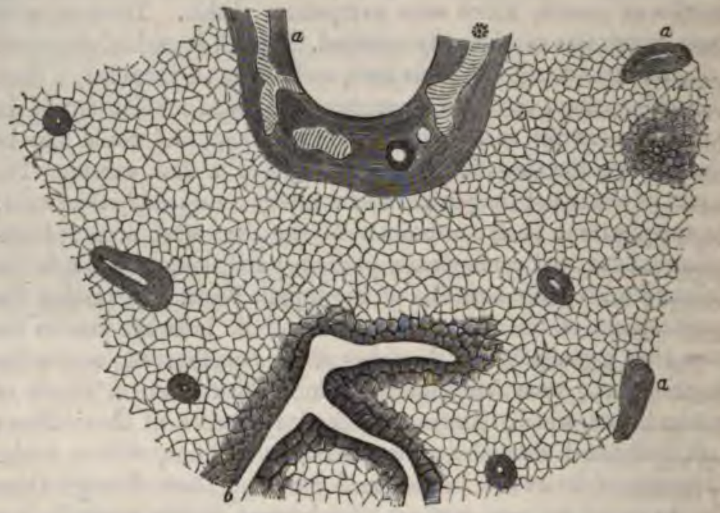

FIG. 26. A magnified thin section of a liver, showing the changes in its structure produced by chronic atrophy. The sheaths of the branches of the portal vein (*a*) are seen to be remarkably thickened, and form a striking contrast to the thin walls of the hepatic vein (*b*). At *, hepatic arteries are represented as contained in the sheath of a branch of the portal vein.

adherent hepatic cells. Further examination proved that the former supposition,—according to which the network was made up of the capillaries, which were partially destroyed and densely aggregated, owing to the disappearance of the hepatic cells,—was, without doubt, the correct one. This supposition was favoured, in the first place, by the circumstance of cells being found in the meshes, and, secondly, by the attempts at injection, by means of which we succeeded, in some places at least, in throwing the injection both from the portal and the hepatic veins into the granulated network of tubes.* In many parts of the liver the injected substance did not enter at all into the capillary system; and even water, when injected, could not be forced through it. Moreover, in the apertures of the larger vessels, the lining membrane at some places could be seen coloured by brown granules, in the same way as the network of small tubes.

The destruction of the hepatic capillaries, which has just been described, as well as the shrivelling, or complete disappearance of the secreting cells, was not uniform throughout the entire organ, but was more advanced at one part than another, as might be shown by the variations in the mode of distribution of the substances injected into the vessels, and still better by an examination of fine sections of the gland. At some places it was scarcely possible to isolate a few stunted cells from the fresh substance of the gland; at other places the cells were more abundant, although for the most part greatly altered in their characters. Most of them were small, pale, and without any granular contents; in only a few could a nucleus be detected; their walls seemed wrinkled and at some places folded, so that their outlines presented a sacculated, angular appearance. Brown-coloured molecules were accumulated in large quantity in some of the cells, while others were completely filled with brownish-black contents, and here and there throughout the tissue of the liver were seen large granules, and little rods of bile-pigment;—morbid appearances which proved that the passage of bile into the excretory ducts had been impeded.

The gall-bladder contained a small quantity of thin, pale-yellow, turbid bile, which, when heated, threw down a coagulum of albumen.

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* The appearances here described are figured in the Atlas (Plate IV., Fig. 4). The yellow substance injected from the portal vein is seen to penetrate only to a short distance, whereas that injected from the hepatic vein advances much farther into the network.—TRANSL.

The transudation of this albumen was owing to the increased pressure exerted by the blood, upon the walls of those capillaries of the portal vein, which remained exempt from the process of destruction.

In this case, the primary cause of the atrophy, of the destruction of the capillaries, and of the disappearance of the secreting cells, depended apparently upon the disease of Glisson's capsule, which, proceeding from the ulcerated spot at the pylorus, extended into the fissure of the liver, and along the course of the portal vein as far as its ramifications. The disease attacked the portal vein itself, as proved by the marked structural changes in the walls of that vessel, the brownish spots upon its inner surface, &c.; in the trunk and larger branches, it gave rise to thickening, paralysis of the muscular tissue, and dilatation; but, in the smaller branches and in the capillaries, to the abnormal conditions just mentioned, obliteration, &c.

I have frequently observed similar changes arise from a cancer of the stomach, upon which cancerous peritonitis has supervened; in these cases, Glisson's capsule has been infiltrated with cancer far into the substance of the liver, and the organ itself has been greatly atrophied.

It is not always, however, that the portal vein in chronic atrophy of the liver becomes enlarged, in the manner above described. I have observed the same appearance in cases where the atrophy of the liver had another origin, directly involving the capillaries, and in which Glisson's capsule was of normal thickness.

Observation No. XXIV.

Tertian and Quotidian Intermittent of three months' duration.—Anasarca.—Ascites.—Diarrhœa.—Death from Exhaustion.

Atrophied Pigment-Liver and Pigment-Spleen.

A boy, 10 years of age, who had been much neglected, and who, for three months had suffered almost uninterruptedly from intermittent fever, at first of a tertian, and afterwards of a quotidian type, came to the Hospital at the beginning of May, 1855, with a pale anæmic countenance, and suffering from profuse watery diarrhœa. The spleen was moderately enlarged. No hepatic dulness could be detected in front; in the axillary line it amounted to 2 centimètres (¾ inch). The lower part of the abdomen contained a considerable quantity of fluid, but there was scarcely any obvious œdema of the feet. The evacuations by stool, which occurred six or eight times in the course of the day, were thin, of a pale greyish-yellow, and without any traces either of blood or dysenteric exudations. Pulse 90, and small; no elevation of temperature. Appetite slight. The urine, when treated with heat and nitric acid, exhibited scarcely any turbidity.

The attempts to check the exhausting evacuations by means of chloride of iron, nux vomica, &c., and to counteract the anæmia by means of a regulated diet, proved fruitless. After three days' stay in the Hospital, the boy died.

Autopsy.

Considerable ascites; slight anasarca. No important changes in the organs of the cranial or thoracic cavities. The mucous membrane of the stomach and intestinal canal pale, at some places œdematous; the serous membrane of the small and large intestine exhibited here and there livid spots,—the remains of ecchymoses.

The kidneys were normal, with the exception of a few isolated pigment-granules in the glomeruli.

The spleen was large, firm, congested, and densely infiltrated with black pigment.

The liver was very small and atrophied; numerous deep depressions were seen upon its outer surface. Its parenchyma was congested, brownish-black, and of tenacious consistence. Yellow size injected into the portal vein passed along very incompletely; in thin sections of the injected tissue, the ramifications of the portal vein as far as their entrance into the lobules, could be seen to be much enlarged, and, at some places, unequally so. Black pigment was observed in the interior of the capillaries, only a few of which were injected; a large number of them appeared to be no longer permeable.

OBSERVATION No. XXV.

Persistent and oft-recurring Quotidian Intermittent.—Hydræmia. —Anasarca.—Ascites.—Profuse Diarrhœa.—Death from Exhaustion.

Atrophy of the Liver.—Blocking-up of the Capillaries with Pigment.

Mrs. M., aged 26, before she came to the Hospital, on April 27th, 1855, had suffered for several months during the winter from intermittent fever. This fever continued for a long time after the patient's admission, and did not permanently disappear until after the repeated administration of large doses of quinine. The patient became gradually reduced to an extreme degree of anæmia, to which general anasarca and ascites were superadded, and which rapidly increased to a critical degree, under the additional operation of persistent and profuse diarrhœa (to check which, the vegetable and metallic astringents, particularly the muriate of iron, were employed without effect). Death took place six weeks after admission. Neither albuminuria, nor derangements of the sensorial functions, which before admission had accompanied the intermittent fever, were observed at any time during the patient's stay in the Hospital.

Autopsy.

There was nothing abnormal in the brain; the lungs were anæmic and collapsed; and there was a moderate amount of effusion in the pleural cavities. The heart was normal. The abdominal cavity contained several pounds of clear serous fluid.

The liver was small;* it weighed 0.7 kilogr. (1 lb. 8⅔ oz. av.), and was thus less than one-half the normal weight; at the same time it appeared shrivelled and tenacious (*welk u. zähe*), and its parenchyma was congested and dark. Under the microscope, numerous flakes of pigment could be seen in its capillary vessels, some of which were remarkably large. The ramifications of the portal vein were greatly enlarged as far as the periphery of the lobules, but attempts to inject them proved very unsuccessful. A large proportion of the capillaries contained pigment, and were, from this cause, impermeable by the injection. The surrounding hepatic cells were either atrophied or filled with fat; at some places, lardaceous matter (*speckstoffe*) was observed. Sugar could not be detected in the liver.

The spleen was small,† firm, and coloured brownish-black, owing to dense accumulations of pigment; its capsule was thickened.

The kidneys were normal, except that isolated pigment granules were seen in the glomeruli. The mucous membrane of the intestine was pale and œdematous; in the sigmoid flexure there were a couple of superficial catarrhal ulcerations.

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* The right lobe was 5½ inches long and 4 broad; the left, 4 by 3¼; the thickness amounted to 2 inches.

† It measured 3 inches in length, 2½ in breadth, and 1½ in thickness Its weight amounted to 0.14 kilogr. (5 oz. avoird.).

The origin of the atrophy in this case could not be traced with certainty. The pigment flakes and granules, which not unfrequently are formed in the spleen in the course of severe intermittents, and from this organ are transmitted into the portal vein, partly pass on through the capillary network of this vessel, and, partly owing to their size, remain firmly impacted in it, and give rise to occlusion ending gradually in obliteration. The obstructed flow of blood entails an arrest of the secretion, and destruction of the glandular cells. Occasionally it happens that large clots are swept along into the vessels of the liver; scar-like depressions are then produced at those parts of the surface of the organ to which the obstructed vessels lead. (*See Diseases of the Portal Vein.*)

Observation No. XXVI.

Atrophy of the Liver with Fatty Infiltration.—Dysenteric Cicatrices.—General Dropsy.

Gottlieb Günther, aged 34, came to the Hospital on October 20th, 1854, on account of catarrh of the stomach and bowels, accompanied with fever; when convalescent, he was seized in the Hospital with a slight attack of dysentery, which at that time was prevalent in the wards. Although the purging was soon checked, still the patient recovered very slowly; he continued anæmic; ascites came on, and anasarca and hydrothorax were superadded. Along with these symptoms, the urine was free from albumen, the heart normal, and there was no disease in the respiratory organs; the appetite was tolerably good; the bowels were regular, and the stools were firm, and only abnormal as regards their colour.

The attempts to counteract the hydræmia by means of quinine, steel, wine, and an easily-digested animal diet, proved fruitless; the dropsy gradually increased to such a degree, that the respiration was greatly impeded. The administration of colocynth, it is true, led to a diminution of the anasarca, but the patient's state of exhaustion rendered it necessary to abstain from its use; diuretics took no effect; the kidneys secreted only a small quantity of dark urine free from albumen. Intercurrent attacks of pulmonary œdema, which ultimately threatened suffocation, were treated with benzoic acid and camphor. In the middle of December, bed-sores appeared; the exhaustion now rapidly increased until the 19th, when death ensued after a protracted agony.

Autopsy.

There was nothing abnormal in the cavity of the cranium. Both lungs were œdematous, and there were about four pounds of clear fluid in the pleural cavities; the heart was normal. The abdominal cavity contained about ten pounds of serum. The stomach was tightly contracted; its mucous membrane was pale, except at the pylorus, where it was darker; the lining membrane of the small intestine was pale, and at some places œdematous, that of the cæcum and of the ascending colon presented similar characters; in the transverse colon, and extending downwards as far as the rectum, there were found numerous black spots, and superficial ulcerations completely cicatrised. In the meso-colon of the sigmoid flexure, there were observed thick, white, radiating bands, like those of a cicatrix, which compressed a portion of the veins passing through them, and tied down this portion of the bowel in front of the spinal column. The mesenteric glands, the pancreas, and also the kidneys were normal.

The spleen was somewhat enlarged, anæmic, of a uniform brown colour and firm.

The outer surface of the liver presented several scar-like depressions; its margins were sharp and furnished with a broad white rim. The size of the organ was very much reduced; its parenchyma was of a dirty brownish-yellow colour, and abnormally soft. The gall-bladder contained a pale-yellow turbid fluid.

Observation No. XXVII.

Chronic Dysentery.—Displacement of the Intestinal Canal.—Atrophy of the Liver.—Death from Exhaustion.

Gottfr. Dräsner, aged 53, a powerful man for his age, who had been obliged to work in the open air during changeable weather, had been suffering for four weeks from diarrhœa, with copious thin stools of a pale greyish-yellow colour, and from abdominal pains, and tenderness upon pressure, along the course of the descending colon, The tongue was slightly coated; the appetite, diminished; the respiratory organs, normal; no cardiac bruit. The size of the liver could not be ascertained; no dulness could be detected anywhere in the hepatic region.

An emetic was administered; and a mixture was ordered containing decoction of calumba and tincture of nux vomica. After a few days, the diarrhœa disappeared; the appetite returned; and the patient was disharged cured on the 14th.

On the 12th of November, Dräsner again presented himself at the Hospital. The diarrhœa had returned soon after his discharge, and had lasted for three weeks uninterruptedly and with great severity. The patient appeared pale and emaciated; the skin was dry and hot; pulse 105 and small; the pains and tenderness in the region of the colon were more severe than formerly; and the stools were of the same thin, greenish-yellow character. Even now no hepatic dulness could be made out, although the abdominal walls were flaccid, and the bowels contained no great accumulation of gas.

The patient took calumba and opium; but on the evening of the 15th he fell into a state of collapse, and became unconscious. He died early on the 16th.

Autopsy.

The body was emaciated, but free from œdema. There was nothing remarkably abnormal in the organs contained in the cranial and thoracic cavities. In the abdominal cavity, the first thing that was observed was a remarkable displacement of the intestinal canal. (Fig. 27.) A portion of the ileum, *d*, was situated where the liver ought to lie, and here it was bounded by the lung, close to the fifth rib; the liver was quite invisible. The first curve of the colon, *a*, lay in the left hypochondrium. The other portion of the intensely injected small intestine—the jejunum, *b*, was superimposed over the descending colon and compressed it. The sigmoid flexure, *c*, lay on the right side, and a long coil of it covered the cæcum. The mucous membrane of the stomach, but only that portion near the pylorus, was softened and livid. The lining membrane of the ileum was pale. In the jejunum, the serous coat was intensely injected, and the mucous membrane presented a small ulcer in process of cicatrisation and loaded with pigment. The cæcum contained pale clay-coloured fæces; the mucous membrane was here livid, and at the commencement of the colon, it was covered with patches of grey exudation, which, still further on, formed a uniform layer. In the sigmoid flexure the lining membrane was reddish-brown, and at some places was infiltrated with exudation; the rectum was normal.

FIG. 27.

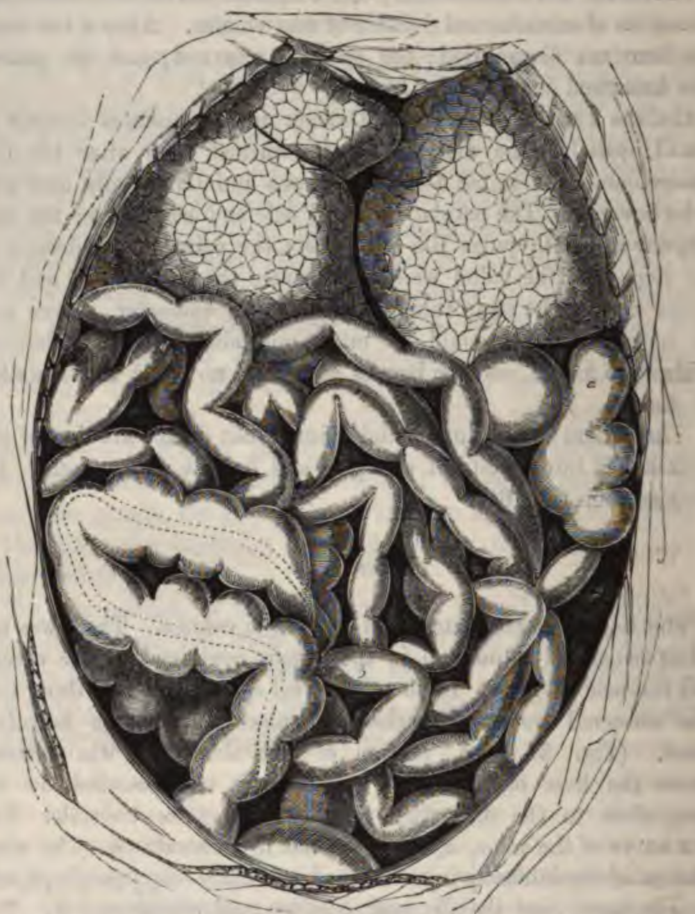

FIG. 27. Arrangement of the abdominal viscera in a case of chronic atrophy of the liver. The liver is completely obscured by the superimposed bowels. There is also great displacement of the bowels, as explained in the text.

The spleen was enlarged, of a uniform reddish-brown colour, and firm consistence; it weighed 0.33 kilogr. (11⅔ oz. avoird.).

The liver was very small, and weighed 1.13 kilogr. (1 lb. 7⅞ oz. av.); its outer surface was smooth, and there was a blank rim (*leere Säume*) along its sharp margins. The parenchyma was congested, and of a uniform reddish-brown colour, without any indication of lobules; the branches, as well as the trunk of the portal vein, were enlarged; but there was no thickening of their sheath. The hepatic cells were small, pale, with sharp, angular outlines, and were destitute of molecular contents; some of them contained dark-brown granules, which not unfrequently completely filled up the cavity of the cell. Injection penetrated very imperfectly into the vessels.

Observation No. XXVIII.

Fibrous thickening of the Mesentery, with firm adhesions of the small Intestine and of the Omentum to the abdominal wall.—Deposit of bluish-black pigment and cicatrizing ulcers in a coil of the small Intestine, three feet long.—Chronic Atrophy of the Liver.—Ascites and General Dropsy.

Wilhelm Leidling, a baker, aged 36, had suffered for years from a right inguinal hernia, which became strangulated on the 15th of March. To have this reduced, he was brought to the Monastery Hospital of the Brothers of Charity, at Breslau, where, for six weeks after the spontaneous reduction of the rupture, he was treated for diarrhœa, accompanied by great debility and abdominal pains. The diarrhœa, which, so far as could be gathered from report, never was accompanied by bloody stools, remained uncured; it exhausted the patient more and more, and led to dropsy, so that at the end of six weeks more, viz., on July 1st, 1856, it became necessary to admit the patient into the clinical ward.

The dropsy had by this time become general; along with great ascites, there was double hydrothorax and extensive anasarca. The heart and lungs were normal; the appetite was slightly impaired, and there were one or two thin, pale-yellow motions daily; the patient complained of tenderness on pressure, and also of dull pains coming on spontaneously, to the right of the umbilicus. The hepatic dulness in the mammary line amounted to 3 centimètres (1¼ inch); the spleen could not be measured, in consequence of the anasarca. The urine was scanty, reddish-yellow, and without albumen. The number of white blood corpuscles, in a specimen of blood obtained by the cupping-glass, was not found to be increased, and hence there was no leukœmia present.

The patient remained in this condition, and no improvement took place, notwithstanding the use of preparations of steel, quinine and other tonics, together with a regulated and easily-digested diet. Although the appetite kept up, and no complications supervened, the evacuations from the bowels continued scanty, and the dropsy gradually increased to an enormous degree, until at last the distended integuments became red and ulcerated; and death took place, after a protracted agony, on July 27th.

Autopsy.

The cranial and thoracic organs presented nothing abnormal, with the exception of a general anæmic condition and an abundant effusion of serum into the pleural cavities. There were also many pounds of clear fluid in the abdominal cavity. The mucous membrane of the stomach was pale. To the right of the umbilicus there was a fold of small intestine, which, together with the omentum, was connected to the abdominal wall by firm adhesions of long standing. The serous membrane of this portion of the bowel was of a dark bluish-grey colour; the deposit of pigment commenced by a defined margin two inches above the adhesion, and extended downwards three feet, where it terminated equally abruptly. The coats of this dark portion of the bowel were intimately adherent and thickened; the corresponding portion of mesentery was indurated and fibrous-looking. The mucous membrane presented numerous irregularly-formed cicatrices, surrounded by dark thickened mucous membrane, similar to those which remain after the cure of a severe attack of dysentery. All the remaining portions of the intestinal canal were perfectly normal. The hernial sac was empty.

The spleen was of its usual size and consistence; its colour was reddish-brown.

The liver was considerably atrophied; its capsule was grey and wrinkled; the entire organ was soft and pliable; its cut surface was homogeneous, of a brown colour, and without any indication of lobules; its consistence was tenacious; the secreting cells were shrivelled up, small, and filled with brown granules; the bile was scanty, pale-yellow, and thin, and contained albumen.

In the uro-poietic system there was nothing abnormal.

In the last three cases (and I could add others of a similar nature), the atrophy of the liver made its appearance along with chronic exudation-processes, and ulcerations of the small and large intestine. These two affections apparently arose from a common origin; although it was not altogether clear what was its nature. There was no chronic peritonitis involving Glisson's capsule. It appears to me, that in these cases there is a similar sort of connection between the hepatic affection and the intestinal disease as exists in some of the hepatic abscesses, which in tropical countries are observed to follow an attack of dysentery. The connecting link is the portal vein, which, according to the nature and manner in which its roots participate in the exudation-processes in the tissue of the intestinal mucous membrane, in one case gives rise to the so-called metastatic abscesses, and in another, produces occlusion of the capillaries, and consequent atrophy of the liver.

The obliteration of the trunk of the portal vein may be followed by similar consequences to those which proceed from destruction of its capillaries within the liver. When the occlusion of the vascular tube is not the result of an attack of acute phlebitis, which is usually accompanied by tumefaction of the liver, the formation of abscesses, &c., it gives rise to a uniform atrophy extending throughout the entire organ. Gintrac* has collected a series of observations, both of simple and cirrhotic atrophy of the liver, in which the portal vein was plugged up. He goes a step too far, however, in regarding occlusion of the vein as invariably the primary and exciting cause, and in attributing even cirrhosis to the same origin. In cirrhosis and, in all probability, in many cases of chronic atrophy also, the coagulation of the blood in the portal vein is manifestly a secondary lesion, depending upon the impermeability of a large number of the hepatic capillaries.

I give here the details of two cases of obliteration of the portal vein, which, even independently of the characters presented by the hepatic parenchyma, have claims to our attention, owing to the nature of their origin and the peculiarity of their symptoms.

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* Observations et recherches sur l'oblitération de la veine-porte. Bordeaux, 1856, p. 39.

OBSERVATION No. XXIX.

Violent Dyspnœa.—Bloody Sputa.—Systolic Bruit in the Pulmonary Artery.—Ascites.—Hæmorrhages from the Stomach and Bowels.—Death from Asphyxia.—Occlusion of the Pulmonary Artery by a Thrombus, in consequence of inflammation of the vessel.—Coagulum of blood in the Portal Vein, Ecchymoses of the Peritoneum, as well as of the Mucous Membrane of the Stomach and Bowels.—Atrophy of the Liver.

Gottfried Schmidt, a joiner, aged 44, was admitted on the 20th of April, 1855.

The man was of an athletic build of body, and only slightly emaciated, but the colour of the skin was pale and waxy; the feet were slightly œdematous; he complained chiefly of debility and dyspnœa. On examination, the thorax was found to be greatly arched; nothing abnormal could be made out in the lungs, with the exception of râles in the lower lobe on the right side. The heart was broader than in the normal state, and, on auscultation, a widely-extended systolic bruit could be heard loudest on the left side, close to the cartilage of the fifth rib; there was no exaggeration of the second sound of the pulmonary artery, and no hypertrophy of the left ventricle. Pulse was 70, and small. The digestive functions were but slightly impaired; the tongue was clean, and the appetite moderate; the stools were of normal character. The hepatic dulness was reduced, amounting to scarcely two centimètres in the sternal line ($\frac{4}{5}$ inch); the spleen was somewhat enlarged; and there was a moderate quantity of fluid in the abdominal cavity. The urine was scanty, dark, and free from albumen.

The disease had been gradually developing itself for a year. About ten months before, dyspnœa and palpitations had appeared, accompanied by hæmoptysis and great debility, without the medical attendant being then able to find any certain proof of the existence of pulmonary tubercles. The pains ceased, but the dyspnœa continued, and the patient's strength did not completely return. The man, however, returned to his work, at which he continued for a long time, until the increasing debility and dyspnœa, and the œdema of the feet, rendered it necessary for him to seek assistance at the Hospital.

He was ordered lactate of iron, and animal diet.

From the 1st of May the dyspnœa increased in a marked degree; the patient complained of great anxiety, and of a feeling of impending suffocation; at the same time there was no impediment to taking a long breath, and everywhere throughout the lungs the vesicular murmur of respiration could be distinctly heard. There was frequent vomiting of a bitter greenish-yellow fluid; the pulse was 110, and very small; the extremities were cool; the countenance was pale, and the features anxious.

On the 3rd of May, increasing dyspnœa and anxiety; repeated vomiting of a greenish fluid, mingled with black flakes; great tenderness of the abdomen; thin, reddish-brown, bloody stools; sudden death at 10 A.M.

Autopsy, May 4th, 24 hours after death.

Skullcap congested; sinuses filled with fluid blood, half-an-ounce of serum at the base of the brain; cerebral membranes moderately congested; substance of the brain anæmic, of normal consistence.

The thyroid gland was somewhat enlarged and congested; the bronchial glands were small and melanotic. The mucous membrane of the trachea, and more particularly of the bronchi, was of a dark livid-red colour, and covered with small whitish distended follicles.

Both lungs were connected to the thoracic walls by somewhat firm ligamentous adhesions; inferiorly they were compressed by a yellow serous effusion in the pleural cavities; they were tolerably congested; anteriorly and superiorly, they were dry; posteriorly and inferiorly, they were œdematous; they contained a quantity of pigment, without any exudation. The heart was large; there were numerous ecchymoses beneath the epicardium; the right ventricle was hypertrophied and dilated, its valves were slightly opaque, and there was some loose coagulum in the right auricle; the walls of the left ventricle were somewhat thinned, but its valves were normal; the aorta was of the ordinary width and its coats were sound.

The pulmonary artery was enlarged, its valves were normal, and the walls of the common trunk were perfectly smooth; near to the place where the main trunk subdivided into the two principal branches, there was a thick, greyish-red, very firm plug, which adhered to the anterior surfaces of the two main branches, but, posteriorly, did not come in contact with the wall of the vessel. The adhesion in front was so firm, that on careful attempts to detach the plug, the lining membrane of the vessel was torn off. The plug presented all the characters of an old clot gradually becoming organized. The walls of the artery at this place were remarkably diseased. The coats of the vessel were visibly separated from one another; and between the inner and the middle coat was deposited a transparent yellowish-grey, very firm exudation, which at some places was more than a line in thickness; where the clot ceased to be adherent, the coats of the artery again came together. Other plugs were found in the ramifications of the pulmonary artery. Some of these, although evidently of old date, lay loosely upon the walls, and could be easily separated from them; the wall at these places was smooth, and the artery was of its usual thickness and free from disease. At other places, again, the clots were firmly adherent, and inseparable, and here the arterial walls presented the same changes as have been described in the main branches, although in a less degree. One of the branches was observed to be obliterated; the opening in the vessel could not be found by means of a probe, and the twig was converted into a solid cylinder.

The endocardium was everywhere smooth; the venæ cavæ were moderately filled with fluid blood, but nowhere exhibited any coagulum or disease of the coats of the vessel; the arteries also were everywhere sound. Thus the coagulum found in the pulmonary artery could have originated in that vessel only.

The œsophagus was pale. In the stomach there was a greenish fluid, mingled with black flakes; the mucous membrane presented a considerable number of recent hæmorrhagic erosions, the surface of which was covered with blood, and the margins infiltrated with the same fluid.

The entire serous membrane of the small intestine and of the mesentery appeared thickly beset with small red ecchymoses. The serum, which was accumulated in large quantity in the abdominal cavity, was reddish-brown and bloody. The mucous membrane of the small intestine, from the duodenum as far as the ileo-colic valve, was of a dirty-red colour, and tumid; the solitary glands were very prominent; the contents of the bowel consisted of bloody mucus. In the large intestine, the mucous membrane was only in a moderate degree coloured and turgid.

As the cause of all these morbid changes in the abdomen, the whole of the blood in the trunk and branches of the portal vein, both in the direction of the intestine, spleen, and stomach, and also in that of the liver, was found firmly coagulated. The coagulum everywhere presented the same characters; it was of a dark reddish-black colour; completely filled up the tube of the vessel, but only adhered loosely to its lining membrane, which was sound throughout. It followed the ramifications of the portal vein deep into the substance of the liver, where it became looser, and no longer completely filled up the tube of the vessel.

The liver was small, shrivelled, and collapsed, of flabby consistence, and of a nutmeg-like structure; the cells were loaded with pigment; the bile was tolerably abundant, and brownish-yellow. There was no sugar present.

The spleen was large; its veins were completely blocked up; its parenchyma was dark and congested, and at many places contained deposits of black pigment. Upon closer examination, reddish-yellow, brown, and black pigment in numerous transition forms could be observed.

The kidneys were anæmic, of normal size and texture; the bladder and prostate were sound.

This case was an illustration of the rare circumstance of an inflammation of the pulmonary artery, which, by the deposit of firm coagula, gradually narrowed the tube of the vessel, and at last produced an almost complete occlusion of both its branches. If any conclusions can be drawn from the symptoms, the affection of the artery commenced so early as ten months before death, at the time when the first indications were observed of a deranged condition of the pulmonary circulation. It was probably at that date that the firm ligamentous obliterations of several of the branches of the pulmonary artery originated. By the narrowing of this vessel the circulation of the venous blood became more and more impeded, until, some days before death, the blood in the portal vein spontaneously coagulated, so as to produce complete obstruction. The sudden appearance of the abdominal tenderness, the vomiting of black flakes, and the bloody evacuations from the bowels, announced the occurrence of this accident during life. The entirely uniform character of the clot, and the fact that there was no disease of the walls of the portal vein, proved that the thrombus did not proceed from disease of the vessel, but was a secondary result of the closure of the pulmonary artery. The absence of any deranged condition in the nutrition of the lungs, arising from the occlusion of the pulmonary artery, was remarkable, when contrasted with the changes in the liver, which result from obliteration of the branches of the portal vein.

Observation No. XXX.

Ulcer of the Duodenum.—Obliteration of the Portal Vein by compression.—Death in consequence of Hæmorrhage from the Stomach and Bowels.—Liver and Spleen of normal size.

A. Petzold, aged 41, a labourer, of robust-build of body, had always been healthy until three years before admission. At this time he suffered for thirteen weeks from dyspeptic symptoms, accompanied by pains in the epigastrium and vomiting of yellow matter.

These derangements in a great measure ceased, and there only remained tenderness in the region of the stomach, coming on in severe paroxysms from time to time. About eight weeks before he came to the Hospital, he was attacked with pains in the fundament, and other symptoms of hæmorrhoids. On the 17th of January, 1858, the patient, who in the morning had eaten his usual food with relish, vomited, while at work, about half-a-quart of dark blood, and for this reason was obliged to take refuge at the Hospital. The hæmorrhage returned twice the same day, but in smaller quantity. On admission, the patient was pale and anæmic, and in a state of exhaustion approaching to syncope; the extremities were cool and the pulse scarcely perceptible. The thoracic organs were normal. The epigastrium was moderately distended, and there was tenderness upon deep pressure above the umbilicus. No induration, however, could be felt. The liver and spleen, as far as could be ascertained by percussion, appeared to be of normal size.

He was ordered ice and solution of alum internally; and cold applications externally.

On the 19th, the vomiting, which had ceased on the 18th, returned twice; there was a tar-like evacuation of the bowels, and the tenderness of the epigastrium had increased.

Was ordered acetate of lead and opium, to continue the ice and the external applications.

On the 20th, vomited twice from 4 to 5 ounces of blood.

On the 21st, the pains had abated, the vomiting had ceased; one bloody stool; syncope; slight delirium; pulse imperceptible; cold extremities; was ordered ether and wine.

On the 22nd, the skin was warmer, and the pulse was again perceptible; one involuntary stool.

On the 24th, delirium; the patient attempted to get out of bed; frequent bloody stools. The collapse increased until death, which took place on the 26th.

Autopsy, 24 *hours after death.*

The membranes of the brain, and the brain itself were moderately congested, and of normal characters; so likewise the bronchi, lungs and organs of circulation presented no disease, if we except a remarkable degree of anæmia. The stomach contained about two pounds of firmly-coagulated blood; its mucous membrane was covered with a thick reddish-yellow tenacious mucus; an inch and a-half from the cardiac orifice, the veins could be observed to be very varicose and enlarged, and filled with firm clots of blood. In other respects, the mucous membrane was pale, of normal thickness, and not ulcerated. In the duodenum, just beyond the pylorus, there was a superficial ulcer, half the size of a silver groschen,[*] and in the middle of this could be seen an opening as large as a pin's head, leading into a sinus, which extended three quarters of an inch towards the median line. This sinus was found surrounded by a thick layer of newly-formed areolar tissue, the cicatrising contraction of which had constricted the ductus choledochus, but had left the vena porta entirely exempt. In the interior of the portal vein, and extending into the right and left branches of this vessel, deep into the substance of the liver, was an accurately fitting thrombus, breaking down in its centre into a cheesy-like substance. Behind the stomach, lay a tumour, the size of a walnut, consisting of a fatty, cheesy-looking mass of exudation, enveloped externally by recent areolar tissue. None of the mesenteric glands were enlarged; no hæmorrhoids could be observed in the rectum; but in the mesentery, immediately below the duodenum, could be seen a melanotic patch, several inches in diameter, the remains of an old extravasation of blood. The mucous membrane of the duodenum and jejunum was pale; that of the ileum was at some places tinged yellow. The cæcum, colon, and rectum contained a large quantity of tarry blood, partly mixed up with solid brown masses of fæces. The mucous membrane itself was everywhere pale and free from ulceration.

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[*] A silver groschen-piece measures about $\frac{3}{4}$ inch in diameter.—TRANSL.

The spleen was anæmic and of normal size; it measured 5 inches in length, 3½ in breadth, and 1⅕ in thickness; its absolute weight was 0.15 kilogr. (5⅓ oz. av.) and its relative weight to that of the body, 1 to 208.

The outer surface of the liver presented numerous white cicatrix-like depressions: its size was not reduced. The right lobe measured transversely, 6 inches, and 7½ inches from behind forwards; the left lobe measured 3½ by 5¾ inches; its thickness amounted to 2½ inches; its weight was 1.90 kilogr. (4 lbs. 3 oz. av.) and its relative weight to that of the body, was 1 to 27.3. Its tissue was anæmic, and finely granular; the hepatic cells were pale, and had few granular contents; a few only contained oil globules; others of them contained molecules of colouring-matter. The bile ducts in the substance of the liver were partially filled with a brown fluid, and a considerable quantity of thick opaque bile was found in the gall-bladder. No sugar was found in the liver. A layer of areolar tissue and fat, of considerable thickness, enveloped the ramifications of the portal vein.

This case is interesting in several points of view. Notwithstanding the complete occlusion of the portal vein, which, judging from the character of the thrombus, must have existed for a long time, there was neither any diminution in the volume of the liver, nor any suspension of its secretion. To what extent this function was impaired it is impossible to determine; we must not, however, conclude that it was in no way affected, from the fact of the gall-bladder being full, inasmuch as the contents of the gall-bladder had all the characters of a secretion which had been stagnating for a long period, being condensed, and containing deposits of cholesterine and particles of colouring-matter.

Gintrac* and Oré,† from similar observations, infer that the secretion of the bile is not due to the portal vein, but to the hepatic artery. Such a supposition I hold to be incorrect, and the more so, as there are observations of an opposite nature, in which the hepatic artery was obliterated, and still the bile continued to be secreted (*Ledieu*). Hence, arguing in this way, one might show that neither the vena porta nor the hepatic artery participated in the secreting functions, or in the nutrition of the liver. The continuance of both processes after obliteration of the trunk of the portal vein, is, in my opinion, dependent upon the intimate anastomosis which subsists between the capillaries of the two systems of vessels, and upon the enlargement which the ramifications of the hepatic artery undergo after destruction of a portion of the portal venous system. One can best convince himself of this by injecting the hepatic artery in a cirrhotic liver, when it will be found, that this vessel presents an unusually abundant and extensive vascular network at those places where the injection has penetrated into some of the capillaries of the portal vein.

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* *Op. cit.*, p. 51.

† Gaz. des Hôpitaux, Sept. 9, 1856.

The absence of enlargement of the spleen, notwithstanding the closure of the portal vein, was accounted for by the profuse hæmorrhages from the stomach.

II. *Symptoms.*

The destruction of a large portion of the hepatic parenchyma in consequence of chronic atrophy, necessarily entails a diminution of the functional value of the gland, which reacts upon the system at large, and the more so, as a series of derangements in the digestive organs take place at the same time, owing to the obstruction of the portal circulation. In this way other functional complaints accompany atrophy of the liver, and make up the clinical features of this disease. The symptoms are developed slowly and insidiously. First, there are derangements of the gastric and intestinal digestion; loss of appetite and a feeling of distention, and tightness at the epigastrium; tongue sometimes clean, and at other times furred; accumulations of flatus in the intestinal canal; and pale greyish-yellow, or sometimes moderately brown stools. The bowels are irregular; constipation and diarrhœa alternate; persistent and profuse diarrhœa is frequently (in 9 out of 18 cases) observed, and soon induces exhaustion; in exceptional cases only are the bowels regular. On examining the hepatic region, the dimensions of the organ are found to be reduced in every direction; sometimes no dulness can be made out at all; in most cases, the organ is quite inaccessible to palpation. The spleen is usually unaltered; in a few cases only (in 7 out of 18 cases) is it enlarged.

Symptoms of defective sanguification and nutrition supervene sooner or later upon these digestive derangements; the patients exhibit a pale cachectic appearance without any jaundiced tinge;

the muscular tissue wastes; and at the same time, as a general rule (in 14 out of 18 cases), accumulations of water take place in the peritoneal sac, which are soon followed by general dropsy. The urine is usually pale and free from bile-pigment; in a few cases it has presented a peculiar hyacinth-red colour; once only has it been observed to become dirty-green when treated with nitric acid.

III. *Mode of Termination.*

Chronic atrophy of the liver, when it reaches an advanced stage, as a general rule, terminates fatally, from the gradually progressing exhaustion, or from general dropsy, or from complications more or less intimately connected with the disease of the liver, such as cancer of the stomach, chronic dysentery, &c. In two cases, death took place by peritonitis, in a third, from delirium tremens, and in a fourth, from profuse bleeding from hæmorrhoids. The disease is usually protracted over many months.

IV. *Diagnosis.*

The diagnosis of chronic atrophy of the liver is not unfrequently attended with difficulties, especially when the patient is not seen until the ascites and the œdema of the abdominal walls are so considerable as to render the examination of the abdominal viscera difficult or impossible. We must be mainly guided by the diminution in the size of the liver, by the obstinate gastro-enteric catarrh, the deficiency of bile in the fæces, the ascites, and the cachexia; these symptoms obtain a diagnostic value, when we can exclude all other causes which might account for the ascites or the dyspeptic symptoms. Simple atrophy can only be distinguished from that which arises from cirrhosis, when we can ascertain by means of palpation, whether the surface of the gland is smooth or granular.

V. *Treatment.*

Treatment never holds out much hope of a favourable termination, after the disease is far advanced; and as this is almost always the case when the diagnosis is clear, the treatment can only be directed against symptoms. First, we should take care that the diet is easy of digestion and nutritious; then, the secreting function of the mucous membrane of the stomach and bowels should be regulated by bitter, aromatic and slightly astringent remedies, such as infusions of the root of calamus aromaticus, rhubarb, and cloves, tincture of rhubarb, compound tincture of cinchona, and the aqueous extract of nux vomica. When there is profuse diarrhœa, powerful vegetable astringents are indicated.

The anæmia must be combated by mild preparations of steel, such as the carbonate and lactate of iron, or better still by small quantities of the waters of Pyrmont or Spa,* or of similar springs.

By such treatment we are best enabled to counteract the impending dropsy. After ascites and anasarca make their appearance, we may have recourse to drastic purgatives or powerful diuretics; but the beneficial effects upon the dropsy which may be expected from these remedies, are almost always surpassed by their other injurious consequences. It is best to limit oneself to the use of a diuretic infusion, in combination with some bitter aromatic substance; and to have recourse to paracentesis, when this becomes necessary from the excessive amount of the ascites.

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* The mineral water of Spa, in Belgium, is a gaseous chalybeate, with a temperature of 50° Fahr. It contains more than three-fourths of its volume of carbonic acid, and rather less than four English grains of solid matter in the imperial pint. These solids are made up of carbonate of iron, carbonate and muriate of soda, lime, and magnesia.

The waters of Pyrmont are of a similar character. (See note to page 121.) —Transl.

CHAPTER VII.

THE FATTY LIVER.

(Die Fettleber: Hepar adiposum.)

DEPOSITS of fat in the tissue of the liver are amongst the most frequent structural changes observed in the organ. When this deposit attains a high degree, we are wont to regard it as a disease, and to designate it by the name of fatty liver, or fatty degeneration of the liver.

All attempts to sketch an accurate history of this anatomical lesion from clinical observations, have proved unsuccessful; fatty liver is met with so frequently on opening the dead body, and all clues to diagnosis during life are so inaccessible, that it is impossible to construct a satisfactory symptomatology of the affection. The remark, bearing upon this point, which Louis* made many years ago in his "Recerches sur la Phthisie," is in many respects still applicable:—"Nous manquons de signes capables de la faire connaître à une époque quelconque de sa durée. En vain, j'ai été au devant des symptômes, qui pourraient lui appartenir, je n'en ai recueilli aucun."

Such being the case, one can understand how it is that very different views are maintained in reference to the pathological importance of fatty infiltration of the liver, and its relations to other morbid processes. It has been the custom to regard a liver abounding in fat as diseased, and care has not been taken to define accurately the boundaries within which the normal fatty constituents of this organ may vary without touching upon the confines of disease. Moreover, sufficient attention has not been devoted to the different causes upon which the various forms of fatty liver depend, or to the distinctions which exist between a liver merely abounding in fat and one which has undergone fatty degeneration.

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* Ed. II., p. 129.

1. *Pathology of Fatty Liver.*

Before we consider the pathology of fatty liver, it is advisable that we should study more carefully the variations in the fatty contents of this organ compatible with good health, and that we should investigate the conditions upon which an accumulation of this substance in the parenchyma of the liver is found to depend.

At certain times and under certain conditions, the secreting cells of the liver are so constantly filled with fat, that the idea is forced upon us that some relation must necessarily exist between the presence of the fat and the due performance of the functions of the organ.

The liver of invertebrate animals, invariably contains fat in abundance, as has already been shown by Schlemm, Karsten, Meckel, Will, Lereboullet, Leydig, and others. Some of the cells are filled with drops of oil, whilst yellow molecules of bile are found in others. Hence, Meckel* believes, that we must distinguish two sorts of cells in the liver, one of which is designed for the separation of bile, the other, for the secretion of fat. This supposition, however, is contradicted by the fact that we can detect transition forms between the cells which contain colouring-matter and those containing fat.

Among the Vertebrata, it is known that some of the fishes, such as the Plagiostoma, the Chimæra, &c., are remarkable for a liver abounding in fat. In Raia clavata, Gadus æglefinus, and also in Psyllium canicula, the hepatic cells during the autumn season contain such a large quantity of oil globules, that the organ presents a greyish-white colour, and resembles a reservoir of fat more than a gland destined to secrete bile. In Gadus æglefinus, the dried liver contains 92.71 per cent. of fat, and only 7.29 per cent. of insoluble matter. In the fœtus, we usually find a large quantity of fine oil globules in the hepatic cells; these are scattered through the cavity of the cell, and do not become confluent into large drops, as frequently happens in the later periods of life. At certain times, the quantity of fat appears to be considerable, whilst at others it is less. E. H. Weber † has made the observation, that in chickens between

* Müller's Archiv, 1846, p. 68.

† Bericht der königl. sächs. Gesellsch. der Wissensch. mathem. physik. Classe, 1850., S. 15.

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the 16th and 19th days of hatching, the yolk was absorbed by the blood-vessels from the yolk sac, and passed into the liver. This organ was then loaded with numerous oil globules, and assumed a yellow colour. Not until the chickens came out of the shell, did the quantity of fat contained in the hepatic cells disappear, and the natural brown colour of the organ become gradually developed. According to the views of Weber and Kölliker,* the absorbed yolk mass serves for the formation of blood corpuscles. Lereboullet,† who observed a large quantity of fat in the hepatic cells of a fœtal rabbit, and in those of a mature human fœtus, thought that this was a condition peculiar to the fœtal period of life; but this is a mistake. I have many times, when an opportunity presented itself, examined the fœtal liver of men and animals, and have frequently, although by no means always, detected numerous oil globules in the secreting cells. It would appear, therefore, that the quantity of the fatty constituents of the liver rises and falls with certain stages of development, and possibly also it is influenced occasionally by pathological conditions.

After birth and in the later periods of life, the fatty contents of the liver are subject to numerous variations, some of the conditions of which are well understood.

The first agency which exerts a decided influence in this respect, is Diet.‡

Magendie, in his investigations into the relative nutritiveness of the several articles of food, ascertained that by feeding dogs exclusively on butter, the liver became very fatty, whilst at the same time the skin assumed an oily character, and volatile fatty acids were secreted by the sebaceous glands. Bidder and Schmidt, and more recently, Laue,§ have published similar observations: so that the fact may be regarded as established.

I have myself performed a series of experiments on dogs, in order to trace the stages of the process, and particularly with the object of ascertaining the length of time that food differing only from that to which the animal has been accustomed, in its being mixed with a quantity of fat, requires to take effect upon the liver.* A small piece of the liver of each animal was first removed through a wound in the abdomen, in order to examine and draw the cells;† the animals were then made to take, along with the food to which they had previously been accustomed, from one-half to a whole ounce of liver oil in the day, after which the changes which the organ underwent were examined from time to time. The results of these investigations may be thus summed up:—Already, after 24 hours, the cells exhibited an increase of their molecular contents; after three days, numerous oil globules could be seen; and after eight days the cavities of the cells appeared completely filled with large and small drops of oil. If we examine the liver of an animal fed in this way, in its entirely fresh state, we find the cells considerably enlarged and distended with fine pulverulent molecules; not until after some time does the finely-distributed matter collect into globules, leaving the remaining contents of the cells clear and transparent. In the following figures (Fig. 28) are represented the changes which take place in the hepatic cells after three (A), and after eight (B) days' feeding.

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* Gewebelehre, S. 580.

† Mémoire sur la structure intime du foie. Paris, 1853, page 43.

‡ This has in truth been known ever since it has been the custom to fatten geese in order to obtain a fatty liver, an artifice which, as Th. Willis, observes, was known to the ancient Romans:—"Olim apud Romanos ars fuit, anserem ita pascere ut hepar in immensum accrescens totum præterea corpus præponderaret." (*Pharmac. ration. sive de medic. operat.*, Sect. II., Cap. II., p. 225.)

§ R. Wagner's Physiol. von Funke, S. 171.

FIG. 28.

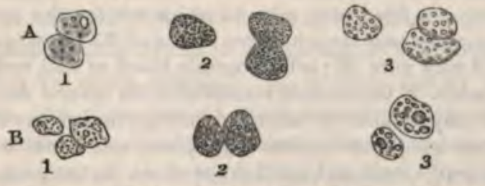

FIG. 28. Secreting cells from the liver of dogs which had been fed upon fat for three (A) and eight (B) days. 1. Their appearance before the commencement of the experiment; 2. Cells, as they appeared after the animals had been fed with oil, in their fresh state; 3. The same, after being kept for some time.

The fatty matter which passes in this way into the parenchyma of the liver‡ disappears again after some time, when the food is changed.

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* Rabbits and frogs are unsuited for this purpose.

† This is necessary, in order to obtain a certain starting-point for the experiment, because the liver in animals is not unfrequently found to be already fatty.

‡ The capillaries of the liver allow fat to pass through them with difficulty, unless it is in a state of very fine division. When olive oil is

How this is effected is still uncertain. It is probable, that when the accumulation of fat is considerable, one portion returns as such to the blood, whilst another is made use of in the formation of bile. It has been often justly observed, that from the chemical properties of the biliary acids, it is probable that fatty matters take part in their original formation. The histological characters of the liver support such a supposition in so far as the quantity of fat diminishes simultaneously with the appearance of yellow matter in the hepatic cells; it is only in exceptional cases that we observe both together.* It can scarcely, however, be doubted, that there are other modes in which the fat is disposed of. The deposit of fat in the liver of the lower animals, and especially in that of fishes, is so abundant, that it cannot be imagined that the substance is entirely consumed in the secretion of bile. In these cases, the gland appears to serve as a depôt for fat which afterwards returns as such into the circulation, and is then subservient to some purpose. In support of this view it is worthy of notice, that even fishes which are destitute of fat in other parts, possess a liver, which is unusually rich in this sub-

injected into the roots of the portal vein, it is retained in the vessels of the liver, and does not reach the right side of the heart. Under such circumstances, there are found in the liver round cysts the size of a linseed, which are filled with oil, and which are for the most part associated in groups of from three to seven; some of the branches of the portal vein are filled with pure oil; others contain blood as well, and occasionally, also firm coagula. If the animals remain alive for several days after the injection, there may be observed scattered through the brown substance of the liver yellow masses containing but little blood, and in which the secreting cells are filled with large and small drops of oil. In one case, in which the animal did not die until after three weeks, the liver was coloured yellow to a great extent, and penetrated by a beautiful dark-red network of vessels. The hepatic cells were distended with oil globules, and in some places they appeared to be crumbling down into a fatty debris. Besides the more prominent yellow portions, there was the normal reddish-brown hepatic tissue, containing cells free from oil. In one case, signs of slight jaundice made their appearance after the injection. Similar changes could not be brought about by injecting the jugular veins with oil.

It cannot be determined whether the fat, upon digestion, is carried directly through the portal vein to the liver; the blood of the portal vein, after feeding on fat, undergoes no change, which would warrant us to answer this question in the affirmative.

[*] Lereboullet even observes (*op. cit.*, p. 86):—"Les cellules graisseuses "ne me paraissent être que transitoires; je crois qu'elles se transforment "elles-mêmes en cellules biliares par dépôt de granules biliares et par "disparition de la graisse, qu'elles renfermaient."

stance, and farther, that according to Leydig, in Paludina vivipara, before the commencement of its winter sleep, the liver is rich in fat, and assumes a white hue, whilst at other times it is brown.

It is not merely food unusually rich in fat that gives rise to these deposits in the liver, but, under certain circumstances, every kind of food, when in too great quantity, has the same effect, even when it is free from fat, and only contains a large quantity of the carbo-hydrogens. Here, however, the deposit does not make its appearance in the liver until the other organs and tissues, such as the cellular tissue, are loaded with fat, nor until the serum of the blood begins to be milky. Lereboullet * observed, that on feeding geese with maize, the relative weight of the liver in proportion to that of the entire body was at the commencement reduced; whilst the cellular tissue first became loaded with fat; not until sometime afterwards did the liver increase disproportionately and become infiltrated with fatty matter; at the same time, the secretion of bile diminished, and the serum of the blood became turbid. In this case, the fat was not conveyed directly to the liver, and its deposit in this organ did not commence until the composition of the blood, and the nutrition were essentially modified in consequence of an improper diet.†

From what has just been stated, it will be understood how a fatty liver is not unfrequently found in individuals who have died suddenly in the bloom of good health.‡ It is then merely a transitory phenomenon, and does not of itself constitute any disease. By a persistence, however, in improper food, the deposit increases more and more, and gradually exceeds the boundaries of health. In such cases there are usually other agencies at work, which aggravate the

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* *Op. cit.*, p. 96.

† Claude Bernard (*Leçons de phys. expérim.*, 1855, p. 149) is of opinion that the sugar which passes from the digestive organs to the liver, is here converted into fat. The milky matter found in a decoction of the liver, and upon the appearance of which, after the use of a diet containing sugar, Bernard mainly relied in making this statement, is poor in fat, and is, moreover, found also in animals which have been restricted for a long time to animal food, and occasionally also in the fœtal liver.

‡ I have found the liver very fatty in a railroad official who was killed at his employment; also in a mason who met with his death from a violent fall; and likewise repeatedly in individuals who have died after a few days' illness, during the eruptive stage of the acute exanthemata, scarlet fever, and measles.

bad effects of diet. First amongst these, is a diminished activity of the metamorphosis of matter, such as is found in individuals who avoid all bodily and mental exertions; secondly, there is a defective action of the liver, from which results an incomplete application to the formation of bile of the fat brought to this organ; and lastly, there are constitutional influences of an unknown nature.

There are some individuals in whom the metamorphosis appears to take place slower than in others; in such persons there is wont to be a predominant tendency to the deposit of fat in the various organs and tissues, whilst the secretion of bile, so far as this can be ascertained, is scanty. This is the case even in animals; and in the experiments of feeding above described, differences were observed in respect to the rapidity with which fat was deposited in the secreting cells, which were of a purely individual nature. These discrepancies apparently depended upon dissimilarities in the development and functional activity of the several organs, which are concerned in the absorption and metamorphosis of the elements of food. Such constitutional peculiarities are hereditary; and this partly explains why it is that a train of abdominal derangements, hæmorrhoids, excessive *embonpoint*, &c., are hereditary in certain families, and how successive generations come to be visitors at Kissingen, Marienbad and Karlsbad. Even as regards the mere digestion of fat, important differences exist. There are individuals who can only tolerate very small quantities of fat, without the functions of the stomach becoming deranged, whilst there are others who consume a large amount without any ill effects, and still continue lean; and again, there are others in whom the use of large quantities of fat is followed by a corresponding increase in bulk.

Along with individual idiosyncrasies, there are agencies of a more general nature which favour the deposit of fat, and with the mode of operation of which we are as yet only partially acquainted; such are, the middle period of life, the female sex, and a temperate, humid, marshy climate.*

The modes of origin of fatty liver hitherto discussed may, for the most part, be reduced to external agencies; in other cases, this condition exists in connexion with internal pathological processes, some of which at least do not at all directly implicate the liver. Of these morbid processes we are particularly familiar with the frequent concurrence of fatty liver with pulmonary tubercle, and other wasting diseases, as also with the drunkard's dyscrasia; both of these morbid conditions, however different they may be in other respects, agree in this, that the blood is usually characterised by the presence of a large quantity of fat, and that a milky turbid serum separates from it. This, indeed, is the cause of the change which the hepatic tissue undergoes in these affections. The production of fatty liver in the course of pulmonary tubercle has been attempted to be explained in various ways; most writers lean to the view which attributes the production of the carbo-hydrogens and fat to obstructed respiration, and to the defective oxydation of the blood resulting from this. Without entirely denying the influence of respiration,—as has often been done, on the ground that in other derangements of the respiratory function, such as emphysema, fatty liver does not make its appearance, and that it is met with in cases where there is tubercular deposit in other parts of the body, such as the bones (*Rokitansky*),—I believe myself, that the cause of this form of fatty liver must be sought for in those changes in the composition of the blood, which take place during the process of wasting. The blood becomes loaded with fat, which is taken up during the advancing emaciation, in order to be employed for supplying the demands of the metamorphosis of matter in the system.* The accumulation of fat in the liver is usually more remarkable in pulmonary tubercle than in other wasting diseases, in which the respiration is unaffected, merely because the impaired absorption of carbonic acid entails a more tedious metamorphosis. In women, in whom the adipose tissue is wont to be more developed than in men, the absorption of fat is greater; fatty liver is, therefore, more common, and more marked in tubercular females than in males.

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* See Prout: On Stomach and Renal Diseases, 5th Ed., p. 255.

The condition of the digestive system exercises a farther influence; the more this is impaired, and the more the secretion of bile becomes from this cause diminished, so much the less of the fat which has been removed from the body is employed in the formation of bile, and so much the more of it accumulates in the liver.

In other wasting diseases, fatty liver occurs certainly less frequently than in pulmonary tubercle, but still oftener than is usually believed. Bright mentions cases in which it appeared in the course of chronic dysentery and along with cancer; Budd records other instances of fatty liver accompanying cancerous ulcers, and I have myself observed the most advanced grade of the disease going along with compression of the spinal cord, in a case where great exhaustion was induced by a sloughing bed-sore. (For further examples, see the Table showing the different diseases in which this affection is met with.)

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* This view was expressed years ago by Larrey, who appealed to the injurious method then prevalent in France, of promoting the formation of a fatty liver in geese. The animals were shut up in close, hot cages, without food of any sort whatever, whereupon they became ill and remarkably emaciated, whilst the liver became enlarged and fatty.

Accumulation in the blood of fat proceeding from another source, is the cause of the fatty liver of drunkards. To what extent the direct influence exerted upon the liver by the alcohol entering the blood, can contribute to the development of the affection in question will have to be discussed in the chapter on cirrhosis.

From what has already been stated, the general conclusion may be drawn, that in every instance in which the blood becomes loaded with fat, either as a consequence of improper diet, or owing to abnormal conditions of the metamorphosis of matter, infiltration of the liver with fat may be developed, either in a transient or permanent form. There are two sets of glands, particularly, which become implicated by this altered composition of the blood, viz.:—the liver and the sebaceous glands of the skin. A greasy, or velvety character of the cutis is thus a frequent accompaniment of fatty liver, and may, under certain circumstances, be of service in diagnosis.

Besides these general influences which act through the medium of the blood, there are other derangements of a local nature, confined to the liver itself, which may tend to fatty degeneration. That such agencies really exist, is shown by the partial development of this affection in isolated masses scattered through the liver, while the remaining portions are comparatively sound. Limited local deposits of this nature are found in atrophied livers and in cirrhosis, where not unfrequently a portion of the lobules, or even the half of an individual lobule, is in a state of fatty degeneration, while the remaining portion or half remains exempt; they are further found in lardaceous (*speckig*) infiltration of the liver, and lastly, in the circumference of cancerous nodules, cicatrices, and inflammatory deposits, &c. Moreover, pale, irregular spots, from ½ to 2 inches in diameter, are not unfrequently observed over the outer surface of the gland, which, in other respects, is normal; these penetrate several lines into the substance of the parenchyma, and then terminate by a defined border. Such places sometimes contain pale, perfectly normal, hepatic cells, but frequently also cells filled with fat. The causes which occasion such local changes, can only be partially investigated with any degree of certainty. In most of the cases, the fundamental causes are abnormal conditions of nutrition, such as are perceived in consequence of hyperæmias arising from an alteration in the character of the fluid pervading the tissues, which are often found in the kidneys, the pulmonary epithelium, the muscles, &c. In this way we must explain the fatty degeneration of the hepatic cells, which takes place in the neighbourhood of inflammatory deposits, cicatrices, and pathological new-growths, and, to some extent, also in cirrhosis. A similar explanation may be given of the formation of fat throughout the entire organ in the cells undergoing disintegration in acute atrophy, and also of the fatty degeneration, which occasionally takes place in the later stages of the so-called lardaceous or waxy infiltration (*der speckigen Infiltration*).

The deposit of fat in the liver which originates in this way, is essentially different in its consequences from the first-described form. In that, the fat was simply deposited in the cells, which were not altered in their other characters; but, on the other hand, in the instances last enumerated, the cells which are infiltrated with an abnormally concentrated plasma, and of which the nutrition is partially impaired, cease entirely to perform their functions. We distinguish this form of fatty degeneration from the former, which we may designate fatty infiltration. In many cases, however, where the deposit of fat is partial, its mode of origin cannot be traced in the manner just mentioned; the same remark is applicable to the deposits of fat, which one often finds in atrophied livers, where some of the cells are collapsed and shrivelled, whilst others are filled with fat, and to the deposits which are found scattered through comparatively sound livers, &c.' In such instances, deranged nutrition may be at one time to blame, and at another time an impaired condition of the secreting function; and it is not impossible that other influences of an unknown nature may also operate.

II. *Structural characters of Fatty Liver.*

So far as my experience extends, the deposit of fat in the liver is always limited to the secreting cells; I have never been able to discover deposits of fat in the intercellular spaces of the parenchyma even in the most advanced stages of the affection.* Appearances are not unfrequently in favour of a deposition in the intercellular spaces, inasmuch as in preparing objects for microscopic examination a number of cells become destroyed, and their fatty contents escape, and thus appear to lie external to the cells. Where, however, the elementary structures are isolated with care, we find the fat everywhere enclosed in cells; these cells are sometimes distended to such a degree that their external membrane only becomes visible after the removal of the fat by the addition of oil of turpentine. In fine sections of the liver made after the organ has been injected and dried, we can see the cells loaded with fat, in the spaces corresponding to the meshes of the blood-vessels; these cells can be freed from their contents by treatment with ether. By boiling the sections with ether, we easily succeed in removing the cells; and then the vessels with their sheaths of areolar tissue alone remain. It is our conviction, that an interlobular fatty liver does not exist, and indeed it would be difficult to reconcile the existence of such a lesion with the anatomical distribution of the elementary tissues of the hepatic parenchyma.

The fat is at first deposited in the form of fine small drops in the interior of the cells, usually close to the nucleus, but also at other parts of the cavity of the cell. These little drops then increase in number and size, they approach nearer to one another, the granular and brown molecular contents diminish, and the nucleus is obscured.

FIG. 29.

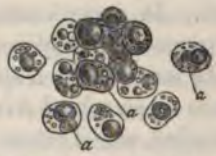

FIG. 29. Hepatic cells, loaded with oil globules, in many of which a nucleus is distinctly visible.

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* J. Vogel (*Icon. Histol.*, Taf. XIX. and XX.), and, more recently, Wedl (*Grundzüge der pathol. Histolog.* Wien, 1854, S. 192; *transl. by the Syd. Soc.*, 1855, p. 162) maintain that they have observed such a deposition of fat. The latter distinguishes two forms of fatty liver; in the one, which he calls the lobular fatty liver, the oil globules lie scattered everywhere through the dirty-yellow substance of the liver; in the second form, or the interlobular, they lie in the interstices of the lobules.

In most cases, the nucleus comes into view on removal of the fat by means of oil of turpentine; it does not usually disappear until the accumulation of fat has become far advanced, and not always even then.*

The little drops of oil afterwards run together, into two, three, or four large drops; and these also, in the further progress of the disease, often become united into one single drop, almost entirely filling up the cavity of the cell. The granular cell contents, with the small oil globules, are then seen to be pressed to one side, and to surround the margin of the large drop of oil like a fringe.

The fat itself in general appears fluid; we rarely see solid granules or drusic masses of crystalline needles of margarine, such as were figured by J. Vogel and Lereboullet.

The accumulation of fat usually causes the cells to enlarge; in cases where the deposit is partial, the cells containing oil, have a larger diameter than other cells from the same liver which contain none; this, however, is by no means always the case. Very small cells containing fat are sometimes found, particularly in atrophy of the liver, and frequently even when the accumulation of fat is considerable, we are unable to detect changes of any sort in the size of the cells.†

In most cases, when the disease is in an advanced stage, the form of the cells becomes altered; their angular outlines disappear, and their form becomes more rounded; when the accumulation of oily matter is considerable, the outer surface of the cell assumes an uneven character from the projecting oil globules. The wall of the cell cannot then always be detected; it does not become distinct until the fat has been removed by means of ether or oil of turpentine; previous to this, it is often impossible to distinguish the cells with their contents from aggregations of globules of oil. In proportion to the increase of the fatty matter, the remaining contents of the cells recede; the fine granular contents diminish, as also the albuminous substance, which is rendered turbid by alcohol, and especially the brown or yellow granules and globules, which a the product of the secreting function. No trace of these substanc is usually to be found in the cells which are loaded with fat, whil they are accumulated in considerable quantity in those which co tain no fat. It is only in exceptional cases that we find fat a pigmentary substances associated. This usually only occurs to a extent, when the deposit of fat has affected all the cells, as far as t centre of the lobules.

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* Lereboullet believes he has ascertained that the nucleus perishes when the deposit of fat reaches an advanced stage. My own experience is at variance with this. Many cells, containing a large quantity of fat, possess a distinct nucleus. (Fig. 29.)

† The cells of a fatty liver measured 0.036 to 0.025 or 0.022 of a line; those of a healthy liver, taken from an individual of the same age, measured 0.030 to 0.022, and another, 0.015 to 0.017 of a line. These numbers indicate a considerable increase of size in the fatty cells.

FIG. 30.

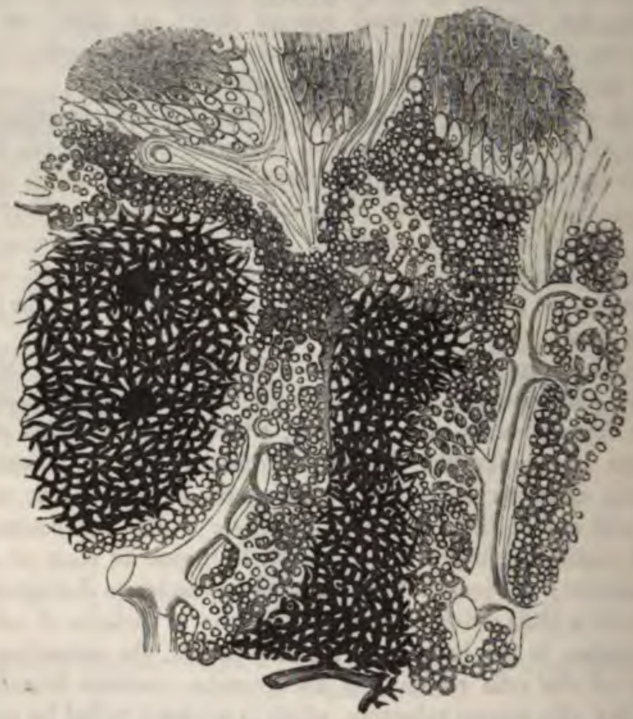

FIG. 30. Represents a magnified thin section of a fatty liver in a moderate degree of advancement. The cells containing oil are seen to be distributed, for the most part, at the periphery of the lobules, in the vicinity of the branches of the portal vein (pale). As the disease advances, they penetrate farther into the substance of the lobules, towards the central veins (dark). A transverse section of one lobule shows that it contains two central veins. The cells containing pigment are situated mostly towards the centre of the lobules, as seen at upper part of cut. Copied from Atlas, Plate VI., Fig. 2.

The alteration which has just been described in the hepatic cells almost always commences at the periphery of the lobules, in the region of the interlobular vessels pertaining to the portal vein,* and advances step by step towards the centre of the lobules, which is supplied by the hepatic veins. (Fig. 30). We can distinguish three stages in the development of fatty liver. In the first stage, the cells in the neighbourhood of the ramifications of the portal vein become fatty, and this abnormal appearance gradually disappears towards their centre, the cells being here partly normal in their characters, but for the most part loaded with pigment.

In the second stage, the deposit of fat advances more than half-way to the centre of the lobules, and it is only in the immediate neighbourhood of the central veins that we can detect cells still containing pigment and free from oil.

In the third stage, the change extends as far as the central vein.†

Some authors, and particularly Wedl, speak of a softening of the fatty liver: a condition in which free fat, molecular masses, and nuclei, are all that remain visible of the hepatic cells, the walls of which must, therefore, have become destroyed. Without wishing to call in question the possibility of such a destruction of the secreting cells, I must observe, that I have never met with this appearance myself in simple fatty liver; on carefully treating the tissue with oil of turpentine, I have invariably detected the walls of the cells still intact. Such a destruction I have only observed at places, where new areolar tissue has been developed in the parenchyma of the liver, in consequence of exudation-processes. In such places we see infiltrated here and there, through the elementary structures of the new tissue, rounded groups of oil globules, and of brown granules, which are the debris of former cells. Here, however, and in all cases where exudation-processes constitute the causes of fatty degeneration, the mode of deposition of fat in the cells, as well as the other characters of these cells, is entirely different from what we find in the ordinary fatty liver of persons labouring under tubercle, and in that of drunkards. In the latter cases, the fat is deposited in the interior of the cells directly from the blood; in the former, the tissue first becomes infiltrated with abnormally concentrated plasma, in consequence of which the endosmotic properties of the cells are altered, and their nutrition is impaired. Under such circumstances the cells are seen in the first place to become filled with a granular albuminous precipitate, obscuring the nucleus, which only becomes visible on the addition of acetic acid; it is not until afterwards that oil globules make their appearance in large number, but even then they are seldom remarkable for their size. This fatty degeneration of the liver, which we believe to differ from fatty infiltration, is usually limited in its extent to the circumference of inflammatory deposits, recent exudations, cicatrices, &c.; it is found extending over the entire organ, apparently as a terminal stage of infiltration of colloid matter, in the so-called lardaceous or waxy liver (*Speckleber*). We have repeatedly met with individuals presenting lardaceous spleen, and in some cases also, lardaceous liver, in whom the liver, which at first had been large and tense, became in the course of treatment smaller, softer, and more shrivelled (*welker*). The hepatic cells were found filled with oil globules and granules, but contained no substance yielding, with iodine and sulphuric acid, the characteristic reaction of colloid matter, which, however, could be detected in the spleen and kidneys.

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* Exceptions to this are rare; in a few cases only I have observed the reverse hold good, the cells at the circumference, next to the portal vein, being free from fat, and those in the neighbourhood of the hepatic veins, infiltrated. In one of these cases there existed a passive congestion of the blood in the roots of the hepatic vein, in consequence of mitral incompetence; another case was an example of hypertrophied fatty liver.

† If we wish to obtain a distinct view of this distribution of the fat, we must inject differently-coloured substances into the portal and hepatic veins, and then cut up the liver into small pieces, and carefully dry it; fine sections of this, made with a razor, may then be examined under the microscope, either at once, or better, after the fat has been removed by boiling with ether. In the latter case, the fatty cells are removed, and the areolar tissue, containing the blood-vessels, only remains.

This degeneration is far more injurious to the functions of the organ than infiltration.*

III. *Quantity of Fat.*

The quantity of fat which is deposited in an advanced stage of fatty liver may be considerable. In one case I found 78.07 per cent. of fat in the substance of the liver after this had been freed of its water;—the quantity of fat, therefore, amounted to nearly four times that of the remaining portions of the tissue;—in its fresh state, this same liver contained 43.84 per cent. of fat, 43.84 of water, and 12.32 of areolar tissue, cells, and blood-vessels. In another case, the quantity of fat amounted to 76.6 per cent. The quantity of water contained in the hepatic tissue is considerably diminished; it falls from 76 per cent. to 50 or even to 43.84.

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* See below: Obs. XXXI.

Along with the fat, which consists of oleine and margarine in varying proportions, with traces of cholesterine, sugar has been found in most cases. Where the deposit of fat has attained an advanced stage, we have also succeeded in finding large quantities of leucine and tyrosine. In some cases, moreover, there is a peculiar yolk-yellow substance, which differs essentially in its characters from bile-pigment. It is worthy of notice, that the decoction of the liver is always found poorer in free acid than in the normal state. Cystine has been sought for, but in vain.

IV. *The General Anatomy of Fatty Liver.*

It is not always possible to arrive at a positive opinion as regards the fatty contents of the liver, by means of simple inspection. Moderate, or even advanced degrees of this abnormal condition do not always alter the colour and consistence of the organ in such a manner, that they can be distinguished with certainty from other changes in the gland, and especially from anæmia; there is a pale and soft liver, which in its characters so closely resembles the fatty liver, that it is liable to be mistaken for it, and yet which contains no fat.

A perfectly certain decision in this matter can only be arrived at by means of the microscope. I do not regard the coating on the blade of the knife which remains after making a section of the organ, and upon which anatomists lay great stress, as a criterion to be relied upon, because this coating is also observed in the case of soft livers containing but little fat, when the cells are only loosened in their adhesions to one another, and contain a quantity of granular matter.

It is true, that forms of fatty liver are met with, which with some experience can be immediately recognised as such; such for example, as the large flattened, dull-yellow liver, with rounded margins, and a smooth,* tense peritoneal covering, which on section is found to contain but little blood, and to present a colour similar to that of autumn foliage (*Rokitansky*). These characters, however, cannot constitute a rule for our guidance, inasmuch as there are so many exceptions to them.

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* The outer surface occasionally appears to be slightly granular, from the peripheral portions of the lobules which contain fat becoming more

The size and weight of fatty liver are liable to great variations; they may exceed, correspond to, or be less than, the normal size and weight. When the results of numerous weights and measurements are taken together, the weight and volume are on the whole increased. Out of 34 cases of fatty liver in adults, the average weight was found to be 1.6 kilgr. (3 lbs. 8½ oz. avoird.) in males, and 1.5 kilgr. (3 lbs. 5 oz.) in females.*

The ratio of the weight of the liver to that of the entire body was as 1 to 28 in males, and as 1 to 25 in females.†

The average measurements were as follows:—

In males, right lobe 5.3 inches; left lobe 3.4 in. transversely.
" " 7.7 " ; " 5.6 " longitudinally.
Thickness, 2.1 inches.

In females, right lobe 5.8 inches; left lobe 3.3 in. transversely.
" " 6.6 " ; " 5.6 " longitudinally.
Thickness, 2.3 inches (Paris inches).

The weight of the spleen in these cases amounted in men to 0.23 kilogr. (8.1129 oz. avoird.); and in women to 0.24 (8.465 oz. avoird.); and the relative weight of the organ to that of the entire body was as 1 to 202, and as 1 to 156.

The relative weight of the spleen to that of the liver, was as 1 to 6.90 in males, and as 1 to 6.25 in females.

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prominent than the central portions. For the relation of this form to granular liver, see chapter on "Cirrhosis."

* Cases, however, have been met with in which fatty liver has attained a great weight; thus, in a female aged 40, it was found to weigh 3.4 kilogr. (7 lbs. 8 oz. av.), and its relative weight to that of the whole body was as 1 to 20.

† Lereboullet has attempted to determine the changes in the relative weight of the liver to that of the entire body which the organ undergoes in geese, from being made fatty. Before the commencement of the feeding on maize, it was as 1 to 26.5; after nine days' feeding, it was as 1 to 30; after fourteen days, as 1 to 18; and after twenty-eight days, as 1 to 12.8. Thus the consequences of excessive feeding manifest themselves during the first nine days, in an increase of the weight of the body; but after this the increase of weight is mainly in the liver.

Besides the hypertrophied fatty livers, we also meet with others, which are atrophied, their size and weight being less than that of the normal state, although the amount of fat which they contain is considerable. Such atrophied forms are by no means rare.

The form of fatty liver is in many cases peculiar; the organ increases mainly in breadth, is flattened out, diminishes in thickness,* and has its margins rounded. But to these characters there are also numerous exceptions; even when the deposit of fat is considerable, the margins of the organ may be sharp, and its measurements in every direction may remain normal.

The colour in general becomes paler, and the more so, in proportion to the abundance of fat, and the extent to which the normal contents of the hepatic cells and the capillary injection are reduced. A considerable amount of fatty deposit may take place without the colour being essentially changed. In most cases, the parenchyma of the liver assumes a reticulated appearance; we find a reddish-brown and a pale-yellow substance alternating with one another; the latter usually forms rings surrounding little rounded islets of the former substance; or the brownish substance presents longitudinal or sometimes leaf-like figures, bounded by a pale margin.† Which of these appearances presents itself depends upon the direction in which the lobules are divided, the latter occurring when the section falls parallel to the branches of the hepatic veins, but the former, when the central veins are divided transversely. The pale parts almost always correspond to the periphery of the lobules, the region of the portal vein, in which the fat is usually first deposited, while the dark parts correspond to the hepatic veins around which the cells are wont to continue rich in pigment, and the capillaries, filled with blood.* The area on section of the individual lobules is enlarged. In the most advanced stages of fatty deposit the liver is pale-yellow, and only small intensely-yellow dots or streaks stand out of a uniformly-coloured ground; these are the pigment cells in the neighbourhood of the central veins. In such cases the finer vessels on the surface of the organ are completely deprived of their blood; but ramifications of enlarged vessels are seen in the serous covering, the same as in cirrhosis. Sometimes we see larger yellow islets scattered through the parenchyma, corresponding to places where there has been a particularly great obstruction to the bile.

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* This is shown by the mean measurements above given.

† The nutmeg-like appearance which the liver assumes under such circumstances becomes the more marked, the greater the deposit of pigment in the cells around the central veins and the larger the quantity of blood contained in these veins.

The same alternation of colours is observed in consequence of obstruction of bile, where the deposit of pigment in the cells always takes place first around the central veins of the lobules.

It is also observed, with somewhat different shades, to be produced by irregularities in the distribution of blood, and by congested states of the roots of the hepatic veins, which are of such frequent occurrence. Hence the nutmeg-like appearance of the cut surface can only be employed for the diagnosis of fatty liver with great caution; we have to distinguish between a fatty, a pigmentary, and a hyperæmic nutmeg liver.

When the deposit of fat does not take place in a uniform manner, it may assume the form of streaks more than an inch in length, or of pale insular masses of a greater or less size, occasionally not exceeding in diameter that of a bean or pea. These patches are more frequently observed upon the surface (from which they penetrate only a few lines into the parenchyma), than deep in the interior of the organ.

The consistence of the liver is wont to diminish in proportion to the increase in the deposit of fat; it becomes flabby, abnormally soft and easily broken up, and, like œdematous areolar tissue, retains impressions made upon it for a long time.

The waxy liver (*Wachsleber*) formerly described by Home, constitutes an apparent exception to the characters just described, being remarkable for its great hardness and firmness. This form of disease usually belongs to the so-called colloid or lardaceous degeneration of the gland; but there are cases in which the cause of the abnormality must be attributed to the deposition of fat of a firm character.† When we compare the ethereal extracts of different fatty livers with one another, no small differences may be observed in respect to the consistence of the fat. No quantitative analysis, however, of the amount of margarine has yet been made.

Lastly, we would mention one other peculiar form of fatty deposit of rare occurrence, viz.:—that which has its seat principally in

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* It is much more difficult than is usually believed to distinguish between the central and peripheral portions of the lobules, inasmuch as very different appearances are produced, according to the direction in which the section of the blood-vessels passes. The matter is only to be decided by injecting different coloured substances into the blood-vessels.

† Laennec, *Traité de l'auscultation*, Tom. VI., p. 36.

Glisson's capsule, and following the course of this and the bloodvessels penetrates deeply into the substance of the liver. I have seen this abnormal condition developed in a high degree in one individual, who died from obliteration of the portal vein. In this case, the trunk of the portal vein, before its entrance into the liver, was closed up by the compression of collections of pus in the surrounding areolar tissue. No fat was observed to be deposited in the secreting cells.

v. *Frequency with which Fatty Liver occurs.*

It will be seen from the previous remarks on the causes of fatty liver, that higher or lower grades of this alteration of structure are not unfrequently met with. In order to obtain accurate and reliable data as to the frequency of occurrence of fatty liver, this gland has been for a long period examined for fat at the autopsies in All Saints' Hospital, and always with the microscope; sufficient information on the point cannot be derived from simple inspection. The results of 466 examinations of this nature are collected in the annexed Table.

Considering the powerful influence exercised by the habit of living, it should be mentioned, that these cases strictly refer only to Breslau, and to the class of the community who are wont to seek refuge in public hospitals; in connection with which we must observe, that hepatic affections generally are of frequent occurrence both here and in the neighbouring Sclavonian provinces. The frequency of fatty liver in other countries can only be determined by direct examination. So far as I can trust my memory, I have seen this affection less frequently in Göttingen than in Holstein and Silesia; it has also appeared to be more frequent in summer than in winter.*

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* As regards the different grades mentioned in the Table, I understand by fatty liver of the highest grade (I.) those forms in which the deposit extends through all the hepatic cells as far as the centres of the lobules. Under the second heading, I reckon those in which the cells are found copiously infiltrated, at least halfway, to the centre of the lobules. It is obvious, however, that we cannot define these different grades with great precision.

TABULAR VIEW OF THE OCCURRENCE OF FATTY DEPOSIT IN THE LIVER, IN DIFFERENT DISEASES, DETERMINED BY MICROSCOPIC EXAMINATION.

	I. Fatty Liver of the highest grade.			II. Cells rich in Fat.			III. Quantity of Fat small.			IV. Cells free from Fat.			Total.			Proportion between the number of the Cases I. and II. and the total number.		
Diseases.	No. of Cases.	Males.	Females.	No. of Cases.	Males.	Females.	No. of Cases.	Males.	Females.	No. of Cases.	Males.	Females.	No. of Cases.	Males.	Females.	In all the Cases.	In the Males.	In the Females.
1. Tubercular Affections - -	17	9	8	62	33	29	34	25	9	4	3	1	117	70	47	1 : 1.48	1 : 1.66	1 : 1.27
2. Pulmonary Emphysema - -	—	—	—	6	2	4	5	3	2	2	1	1	13	6	7	1 : 2.16	1 : 3.00	1 : 1.75
3. Pneumonia -	—	—	—	8	3	5	20	12	8	14	10	4	42	25	17	1 : 5.25	1 : 8.33	1 : 3.40
4. Pleurisy - -	1	1	—	—	—	—	4	2	2	1	1	—	6	4	2	1 : 6.00	1 : 4.00	—
5. Heart Disease -	—	—	—	10	2	8	16	10	6	9	5	4	35	17	18	1 : 3.50	1 : 8.50	1 : 2.25
6. Bright's Disease	1	1	—	8	6	2	7	5	2	3	3	—	19	15	4	1 : 2.11	1 : 2.14	1 : 2.00
7. Typhus - -	1	1	—	6	3	3	21	12	9	16	13	3	44	29	15	1 : 6.28	1 : 7.25	1 : 5.00
8. Pyæmia - -	3	2	1	3	2	1	5	5	—	2	—	2	13	9	4	1 : 2.16	1 : 2.25	1 : 2.00
9. Variola - -	1	—	1	7	4	3	14	10	4	7	5	2	29	19	10	1 : 3.62	1 : 4.75	1 : 2.50
10. Intermittent Fever and its Sequelæ	—	—	—	6	4	2	3	3	—	3	—	3	12	7	5	1 : 2.00	1 : 1.75	1 : 2.50
11. Diabetes - -	—	—	—	—	—	—	1	1	—	4	3	1	5	4	1	—	—	—
12. Anæmia and Inanition from Hæmorrhage, Ulcer of the Stomach, Stricture of the Œsophagus, &c.	—	—	—	5	3	2	3	—	3	2	2	—	10	5	5	1 : 2.00	1 : 1.66	1 : 2.50
13. Senile Marasmus, Apoplexy -	1	1	—	8	1	7	8	3	5	2	1	1	19	6	13	1 : 2.11	1 : 3.00	1 : 1.85
14. Cancer - -	—	—	—	2	1	1	11	4	7	8	2	6	21	7	14	1:10.50	1 : 7.00	1:14.00
15. Delirium tremens	—	—	—	7	5	2	5	4	1	2	2	—	14	11	3	1 : 2.00	1 : 2.20	1 : 1.50
16. Constitutional Syphlis - -	—	—	—	6	3	3	2	2	—	—	—	—	8	5	3	1 : 1.33	1 : 1.66	1 : 1.00
17. Chronic Atrophy of Liver - -	1	1	—	2	1	1	2	2	—	2	1	1	7	5	2	1 : 2.33	1 : 2.50	1 : 2.00
18. Lardaceous Liver in the stage of Colloid infiltration - - -	—	—	—	3	2	1	—	—	—	1	1	—	4	3	1	1 : 1.33	1 : 1.33	1 : 1.00
19. Cirrhosis of Liver	2	1	1	7	4	3	4	3	1	—	—	—	13	8	5	1 : 1.44	1 : 1.60	1 : 1.25
20. Obstruction of Bile	—	—	—	1	—	1	4	2	2	4	3	1	9	5	4	1 : 9.00	—	1 : 4.00
21. Cancer of the Liver	—	—	—	—	—	—	6	3	3	3	2	1	9	5	4	—	—	—
22. Individuals who have died suddenly without previous disease -	—	—	—	2	1	1	4	3	1	2	1	1	8	5	3	1 : 4.00	1 : 5.00	1 : 3.00
23. New-born Infants and Children during the first weeks of life - -	—	—	—	5	3	2	3	1	2	1	1	—	9	5	4	1 : 1.80	1 : 1.66	1 : 2.00
Total - -	28	17	11	164	83	81	182	115	67	92	60	32	466	275	191	1 : 3.02	1 : 3.57	1 : 2.25

It appears, then, from this Table, that out of 466 bodies, fatty liver attained its highest grade in 28, or in 1 out of 16.6 cases; and that the hepatic cells were found rich in oil in 164 cases, or in about one-third.* Females are more frequently affected than males with this morbid change in the parenchyma of the liver, the proportion in the latter sex being as 1 to 3.5, and in the former as 1 to 2.2. In healthy individuals, who have died suddenly from accident, the proportion was 1 to 4: in men, 1 to 5, and in women, 1 to 3. Under certain physiological conditions, there is an increased tendency to the deposit of fat in the liver, as in newly-born infants and in children, during the first few weeks of life, where the proportion was 1 to 1.18. In infants who continue taking the breast until death, the hepatic cells are usually rich in fat, as they likewise are in sucking animals.† In pregnant and puerperal females also we very frequently find fatty deposits in the liver.

Among the pathological conditions which influence the development of fatty liver, tubercle of the lungs stands first; of 117 cases of this affection, in 17 there was the highest grade of infiltration, and in 62 the hepatic cells contained much oil. The proportion was as 1 to 1.48: in males, as 1 to 1.66, and in females, as 1 to 1.27.

Next in order comes the drunkard's dyscrasia. Of 13 individuals who died of delirium tremens, in 6 the liver was very fatty, in 3 the organ contained little fat, and in 2, none at all; lastly, 2 died of cirrhosis of the liver.

Constitutional syphilis was in 8 cases always accompanied by a more or less fatty liver; three times the infiltration attained a high degree, and twice lardaceous matter *(Speckstoffe)* could be detected.

When we compare other pulmonary diseases with tubercle, the ratio is found to be entirely different; in pnemonia it was as 1:52; in pleurisy, 1:6; in emphysema of the lungs, 1:2.1. But of 35 observations of heart disease, the frequency of fatty liver was as 1 to 3.5. Fatty liver made its appearance more frequently with Bright's disease (1:2.1) and with intermittent fever and its seqeulæ (1:2). It was an equally frequent accompaniment of senile marasmus, and of inanition from hæmorrhage, diseases of the stomach, &c.

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* This proportion must, as a general rule, be somewhat too great, because, of the 466 cases, 117 were tubercular subjects, who are remarkable for the frequency with which fatty liver is met with in them.

† This circumstance has been already noted by Gluge *(Atlas der path. Anat*, Lief. 1); and more recently by Kölliker (*Verhandl. der physik. med. Gesellschaft in Würzburg*, Bd. VII., Heft 2).

The comparative frequency of fatty liver in the cancerous cachexia (1:10.5) was remarkably small, when contrasted with that observed in tubercle (1:1.4).

Fatty liver of the highest grade was repeatedly observed in acute diseases of a general nature, such as typhus, variola, and pyæmia; of three cases of erysipelas ambulans, this condition of the liver was met with in all.

Among the local diseases of the liver, cirrhosis and the lardaceous liver (*Speckleber*) are remarkable for the amount of fat which they contain. The smallest quantity of fat in the liver occurred in diabetes mellitus. In several cases of stricture of the œsophagus, leading to prolonged abstinence, every trace of fat had disappeared; this was also the case in an individual who had been bedridden for years on account of hemiplegia;—a proof that bodily rest does not alone suffice for the production of fatty liver.

When we compare these results with the observations of Louis and others, we find that there are important discrepancies. Louis found, out of 230 individuals who died of acute and chronic diseases, excepting pulmonary tubercle, that fatty liver only existed in 9, and of these 9 there were 7 of a tubercular diathesis; whilst out of 120 persons who had died of phthisis, 40 had fatty liver. Home, in Edinburgh, observed fatty liver 15 times out of 65 phthisical persons, and these 15 were exclusively females. I believe, myself, that these remarkable discrepancies must mainly depend upon the fact that, without the assistance of the microscope, it is impossible to form a correct opinion as to the amount of fatty matter contained in the liver; moreover, a certain amount of influence must be exerted by the different modes of living, as also by the nature of the treatment to which the patient has been subjected, as for example, whether cod-liver oil has been used or not.

VI. *Pathological Importance of Fatty Liver.*

Various opinions have been entertained as to the nature of fatty liver, and as to its effects upon the entire organism, according to the manner in which it has been supposed to originate. Most observers have classified this structural change with those derangements of nutrition which are wont to be included under the name of atrophy. Andral,[*] Thomson, Barlow,[†] Cruveilhier,[‡] Wedl, Henoch, and others, are all influenced by this view of the matter in their opinions concerning fatty liver, although they differ as regards the more remote causes of the atrophy. Henoch [§] speaks of the deposit of fat in the hepatic cells as a degeneration of the epithelium and other elementary tissues, which takes place in consequence of congestion and exudation. Lereboullet [||] blames a defective organic combustion as the cause of the impaired nutrition of the hepatic cells.

There are unquestionably forms in which the fatty degeneration must be regarded as resulting from a deranged nutrition of the secreting cells. The appearance of the fat is here preceded by an infiltration of the cells with an abnormal plasma, or by some other derangement of their nutrition, which gives rise to a retrograde development, and to the transformation of the cell contents into fat. When this process has terminated, the cells almost always lose their secreting functions. But in by far the larger number of cases of fatty liver, this explanation is inapplicable. The fat is taken up as such into the cells from without; this imbibition is intimately associated with the functional activity of these structures; it rises and falls according to the nature of the food, the quantity of fat contained in the blood, the more or less active nature of the secretion of the gland, &c. The liver serves here as a temporary reservoir for the surplus fat which has been absorbed; functional derangements do not ensue until the overloading of the cells with fat, by compressing their other contents, and by impeding the circulation in the portal vein, begins to affect the metamorphosis of matter in the cells and to interfere with the secreting function of the gland. All these injurious results disappear as soon as the chief portion of the fat deposited is reabsorbed. It is imposs to define accurately this physiological change in the structure of liver from one which would amount to disease.

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[*] Clinique Médicale, Tom. II., p. 240.

[†] On Fatty Degeneration, 1853, p. 9:—"This organ degenerates from general atrophy."

[‡] *Anat. Path. Génér.*, Tom. III., p. 292. Paris, 1856. "La métamorphose "adipeuse du foie n'est donc autre chose qu'une atrophie adipeuse, bien "qu'elle s'accompagne constamment d'une augmentation de volume de cet "organe."

[§] *Klinik der Unterleibskrankheiten*, Bd. I., S. 122.

[||] *Op. cit.*, p. 112. "Le développement de la graisse dans ces cellules "paraît étroitement lié à un ralentissement dans le travail nutritif, et à "la combustion organique, qui est la première condition de ce travail."

VII. *Effects of the deposited fat upon the function of the Liver, upon the entire System.—Symptoms of Fatty Liver.*

The conditions of the system under which fatty liver is met w and the relations of fat to the due performance of the functions of liver, show that moderate accumulations of this substance may e without giving rise to pathological derangements. We should rat regard as a sign of disease, a complete absence of oil globules in hepatic cells, such as is particularly the case in diabetes mellit The deposits arising from the absorption of food, come and go, cording to the nature of the food, and the consequent amount of contained in the blood. They only attain a high grade, when th have been brought on by a long continuance of improper hab of life.

The forms of fatty liver which depend upon consumptive diseas the drunkard's dyscrasia, and other more radical changes in t composition of the blood, or which originate from local changes the parenchyma of the liver, impairing the nutrition or the functions the cells, are more persistent in their duration. These forms, the fore, more frequently arrive at such a high grade as to impair t functions of the organ.

Considerable accumulations of fat in the hepatic cells deran the functions of the liver in many ways; but it is only in rare cas that they suspend it entirely, or impair it to such an extent, th the entire system suffers.

In the first place, they act mechanically by impeding the flow blood through the portal vein, and the excretion of the bile.

Owing to the cells becoming distended with fat, the vascul meshes in which they lie become enlarged, and the calibre of th capillaries is in a corresponding degree narrowed. Hence, as rule, the advanced stage of fatty liver is found to present an anæm aspect.* The circulation, however, is never impeded to any remar able extent, inasmuch as the soft fluid fat appears to yield before the pressure of the blood; the vessels in a fatty liver are usually injected with ease. The obstruction to the circulation of blood is very seldom so great as to give rise to dropsical effusions of any amount; it is, however, sufficient to produce chronic congestion of the gastro-intestinal mucous membrane, which, from slight causes, may become exaggerated into catarrhs, derangements of digestion, diarrhœa, and hæmorrhoids. The enlarged vessels which are frequently seen running through the serous coat in a similar manner as in cirrhosis, prove that the flow of blood may become obstructed in fatty liver of a high grade. Enlargements of the spleen, however, are rare; this organ remains of its medium size, and is smaller on an average than in the case of the lardaceous liver and in cirrhosis. (See Table IV., page 26.)

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* Even the sections of the larger vessels, seen upon a cut surface, n unfrequently, as Cruveilhier has already observed, present an angular place of a rounded outline, in consequence of the unequal lateral compre sion of the swollen glandular tissue.

Besides the impeded circulation of blood, we constantly observe, that the excretion of bile becomes obstructed. The hepatic cells in the vicinity of the central veins are always loaded with pigment, they contain a large number of yellow granules, and not unfrequently are infiltrated with a brownish or yellowish homogeneous matter. The same cause which compresses the capillaries also impedes the entrance of the bile into the commencement of the excretory ducts at the periphery of the lobules.* This derangement also seldom exceeds a certain point; it never reaches such a degree as to give rise to an intensely jaundiced hue of the skin.

In simple fatty infiltration of the gland, it is rare for the functions of the liver—in so far as these influence the metamorphosis of matter, and manifest themselves in the secretion of bile, and in the composition of the blood—to be deranged to any great extent, although there are anomalous cases which must not mislead us. Symptoms of advanced anæmia or hydræmia, such as are observed in lardaceous degeneration and in cirrhosis of the liver, are here only met with in exceptional cases; we must not, however, overlook the fact that pasty-looking individuals afflicted with fatty liver, bear losses of blood and other debilitating agencies badly.

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* Occasionally the larger ducts also appear to be compressed. Under such circumstances, I have repeatedly observed, in advanced stages of fatty liver, sacculated enlargements of the ducts filled with thickened bile. In one case, numerous crystals of hæmatoidine developed themselves in the decomposed bile contained in such a sac. Cruveilhier describes a similar enlargement of the ducts in a fatty liver (*Anat. Pathol. Génér.*, Tom. III., p. 230).

Out of a large number of instances in which the parenchyma of the liver was examined for sugar, this substance was found in most cases, even when the deposit of fat was very considerable; occasionally it was absent, as happens even in the case of the healthy gland, whenever death has been preceded by a disease which entails prolonged abstinence. Thus the accumulation of fat in the liver does not arrest those processes which give rise to the formation of sugar. There can be no doubt, however, that the metamorphoses of matter which take place in the interior of the cells, are altered and impaired by the presence of fat in place of the other contents of these cells. This opinion is favoured by the feebly acid character of a decoction of the liver, and by the presence of abundance of leucine. Additional evidence as to the modifications of the metamorphosis of matter resulting from fatty liver may be expected from further examinations.

It is a difficult matter during life, to judge of the quantity of bile secreted. The colour of the fæces furnishes only an uncertain indication of the quantity of this secretion which is formed within a fixed period of time. The colour may be but slightly altered, even in cases where there is a very remarkable diminution, or a complete cessation of the secretion. In the slighter deviations from the normal amount of bile, there is no corresponding alteration in the colour of the fæces, and the less so, as the latter is influenced not merely by the quantity of bile, but also by the activity of the intestinal absorption, and by the length of time that the bile is retained in the intestinal canal. When we consider the small quantity of yellow matter which is contained in the hepatic cells (which is the more remarkable, the greater the deposit of fat), and when we further consider the above-mentioned alterations in the chemical characters of the gland, as also the experiments of Bidder and Schmidt, who found that the infiltration of the hepatic parenchyma with fat, consequent upon feeding animals with fatty food, reduced the secretion of bile to the same condition as in starving animals, we are justified in assuming a diminution of the secretion as a result of fatty liver. The *post-mortem* appearances also, which are found in cases of very advanced fatty degeneration, coincide with such an opinion. In two cases of this nature, I have found the bile ducts empty, and covered with a greyish-yellow mucus; the fæcal matter in the small and large intestine was ash-coloured. A complete cessation of the secretion, however, is rare.*

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* The statements of Lereboullet (*op. cit.*, p. 100), who observed the

I have not been able to detect any important changes in the quality of the bile secreted in a fatty liver. It only presents those variations of colour and consistence which are dependent upon a greater or less degree of concentration, a shorter or longer retardation in the gall-bladder, and the secreting activity of the mucous lining of the bile ducts. It may be pale and thin, or at another time dark, thick and viscid. Albumen, which was found by Thénard in five out of six cases, I have never met with; neither have I ever observed the large quantity of oil mentioned by Lereboullet.

Addison* has remarked, that the bile is characterised by a peculiar odour, which is developed upon the addition of acids. This, he says, is perhaps the most disagreeable and intolerable odour which is ever emitted by animal matter and can be compared to no other. I have never myself been able to perceive any increase of that peculiar odour, reminding one of fæcal matter, which is exhibited by thickened bile. The opinion, therefore, which seemed to me a probable one, that under such circumstances, a stinking fluid fatty acid might be present in the bile, is not confirmed.†

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characters of the bile in geese, which had been fed with the object of producing fatty livers, have an important bearing upon this matter. After nine days' feeding, the gall-bladder was distended with green bile, containing a moderate quantity of oil globules. After 28 days, however, it was small and puckered, and contained a very small quantity of oleaginous bile, in which flakes of mucus and numerous oil globules could be detected.

* Guy's Hosp. Reports, I., p. 478.

† I have repeatedly determined the amount of oil which is contained in the secretion of the gland, when its tissue is infiltrated to a great degree. The ethereal extract of the dried bile in no case exceeded 0.50 per cent., usually it amounted to from 0.33 to 0.38 per cent., the solid residuum being from 13.1 to 33.6 per cent. of the entire quantity of bile. The bile was of acid reaction, and contained an oily fat along with cholesterine. In many cases, the ethereal extract presented a pale, blood-red colour, and, upon evaporation, left behind a peculiar crystalline colouring-matter. This consisted of reddish-yellow lancet-shaped leaflets, which were partly isolated and partly united in arborescent groups. Along with these, an amorphous pigment, presenting, upon exposure to the air, the play of colours characteristic of chromogen, was not unfrequently present. I have not yet succeeded in collecting a quantity of the crystals sufficient for analysis. (These have since been ascertained by Valentin to be crystals of hæmatine, and not at all peculiar to the bile of fatty liver. See Preface.—Transl.) Moreover, they are by no means peculiar to the bile of fatty liver; they have not unfrequently been absent in it, and

VIII. *Diagnosis.*

From what has been above stated, it will be understood, that the symptoms which accompany fatty liver, and which in practice must furnish the data necessary for diagnosis, are usually of a very uncertain character, and from the nature of the subject must still remain so. The slighter forms of the disease, which can scarcely be regarded as of a pathological nature, give rise to no remarkable derangements; in the more advanced grades of the affection, the symptoms are of such a nature that they can only, under favourable circumstances, be appealed to with confidence. The first which is of service, in a diagnostic point of view, is the alteration in size and form which the liver is wont to undergo when the accumulation of fat is considerable. The diameter of the gland from behind forwards increases, and at the same time the organ becomes flabby and shrivelled (*welk*), bends upon itself and sinks downwards:—two conditions which equally contribute to increase the extent of the dull percussion sound, and to remove the anterior margin to a greater or less depth below the margin of the false ribs. Owing to this sinking of the anterior margin, a fatty liver upon examination appears to be larger than it really is. When the abdominal parietes are soft, and not too thick to permit of palpation being practised with effect, we can feel the rounded margin of the liver, and also make out its soft consistence. The positive value of these signs is not slight, but their absence is altogether without importance, inasmuch as these changes in the size and form of the gland are by no means constant. A second indication for the diagnosis of fatty liver is furnished by the derangements which arise from the interruption of the circulation through the portal vein. The complex train of symptoms so frequently described by the older authors, under the title of 'abdominal plethora,' and which occur in incipient cirrhosis, and in other mechanical obstructions to the flow of blood through the portal vein, are met with here in a more or less marked form:—such symptoms, for example, as an impaired gastric and intestinal digestion, associated with the development of

have been observed under other conditions, as, for instance, in cholera. The colouring-matter of the bile of fatty liver, in other respects, presents the usual characters. In one case, hydrochloric acid produced the same play of colours as nitric acid.

gases, distention and tenderness of the epigastric region, irregular, and usually constipated bowels, a hypochondriacal frame of mind, hæmorrhoids, &c. The fæces are at one time dark, at another, pale and clay-coloured; occasionally we observe in cases of fatty liver an undue tendency to diarrhœa,—a circumstance which was noted years ago by Schönlein. I have had repeated opportunities of examining the bodies of individuals, who during life have had a pasty complexion, who had been wont to suffer from profuse diarrhœa from trifling causes, and in whom death has taken place suddenly from apoplexy, or, in one case, from acute pulmonary œdema, and have found nothing abnormal in the abdomen, with the exception of a fatty liver in an advanced stage. Similar observations have been repeatedly made at the *post-mortem* examinations in hospital practice. In the same way, we frequently find fatty liver in overfed children, who die from exhaustion after profuse pale diarrhœa.*

The skin has been found by Addison altered in such a manner, that were the change constant, it would be of great value in diagnosis. It was pale, anæmic, semitransparent, and waxy, and at the same time soft and smooth to the feel, like satin. The paleness was at one time devoid of colour, and at another dirty-yellow; it was most distinctly marked in the skin of the face, but was also present in other localities. Such changes of the skin are not unfrequently observed in females labouring under tubercle (I have frequently seen them myself), where of course fatty liver was present, as it usually is in pulmonary phthisis; but I am unable to state whether the liver is the cause of the change, or the hectic fever with its profuse sweating; at all events, fatty liver of an advanced grade is met with, without the skin presenting this character. In drunkards with fatty liver, the skin has not unfrequently a greasy, fatty feel; owing to the abundance of fat contained in the blood, the cutaneous secretion becomes loaded with fatty matter, in a similar manner to what takes place from the continued use of large doses of cod-liver oil (where the odour of the cod oil is transmitted through the skin by fatty acids in a fluid state), and to what was observed in dogs fed by Magendie upon butter.

A due regard to the recognised causes of fatty liver is of greater importance in diagnosis. Where an enlargement of the liver is detected in persons labouring under tubercle, in drunkards, or in individuals of a torpid luxurious habit of life, and when this is associated with the consequences which are known to result from obstructed circulation in the portal vein, then the individual symptoms acquire a greater weight than under other circumstances would belong to them.

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* Legendre also has observed diarrhœa in children, in connection with fatty liver; and Bright has connected both diarrhœa and amenorrhœa with it.

Cases, however, are met with, where the derangements to which fatty liver gives rise, are of a much more marked character; the secretion of bile becomes more and more diminished, extreme anæmia comes on, until at length death supervenes under symptoms of increasing exhaustion, or of complete acholia. This train of symptoms is very seldom observed, and only in the excessive grades of fatty infiltration (See Observation No. XXII., p. 245); it occurs more frequently with the fatty degeneration of the gland, which takes place after the parenchyma becomes infiltrated with colloid and other exudations, sometimes along with lardaceous spleen, and at other times independently of this. The organ, which at first is enlarged, is gradually reduced to even below its normal size; its outer surface continues smooth, or becomes slightly granular, the bilious colour of the stools becomes paler, and the appearance of the patient assumes more and more the characters of general cachexia. Under such circumstances, the liver is found to be of normal size, or somewhat smaller, its cells are filled with fat or albuminous granules, the parenchyma is sometimes traversed by bands of newly-formed areolar tissue, and the bile ducts contain but little secretion. Frequently, but by no means always, the parenchyma of the spleen, or kidneys, or sometimes of both organs, are infiltrated with lardaceous matter, not a trace of which can be found in the liver.

When other important organs, such as the spleen, lymphatic glands, and kidneys, are found diseased as well as the liver, it is a difficult matter to determine the share which the liver takes in the generation of the symptoms of general derangement, the cachexia, hydræmia, &c. In many cases, however, the kidneys and lymphatic glands are free from disease, and the spleen is only changed to such a slight extent, that the symptoms must be mainly attributed to the fatty degeneration of the liver.

The diagnosis of fatty degeneration during life is often attended with great difficulties, especially when one has not the opportunity of tracing the entire development of the process. The following symptoms are often of great service for this purpose:—the diminution in volume of a primarily enlarged liver, along with increasing cachexia, a smooth surface of the gland, the co-existence of chronic enlargement of the spleen, the decrease of the biliary secretion, and the presence of those predisposing causes which experience shows to give rise to the colloid and lardaceous infiltration of the gland, such as intermittent fever, constitutional syphilis, diseases of the bones, &c.

I shall only record here one instance of this nature, but, for additional illustrations, I refer to the chapter on Lardaceous Liver (*Speckleber*).

Observation No. XXXI.

Persistent Intermittent Fever.—Anæmia and Hydræmia.—Exhausting Diarrhœa, with but little Bile in the Stools.—Death under Cerebral Symptoms.—Fatty Degeneration of the Liver.—Small Lardaceous Spleen.—A limited Cancerous Ulcer in the Cæcum.

J. Pallifka, aged 31, during July and August 1853, was treated in the Clinique for anæmia without benefit. He was a large, broad-shouldered man, of a pale, waxy countenance, and complained of remarkable weakness, accompanied by wandering pains and deranged digestion. At the origin of the aorta, there was heard a systolic bruit, which was propagated into the carotids; lungs healthy; spleen and liver of normal size; the tongue slightly coated; the bowels were moved two or three times in the day, and the stools were of a pale colour; urine had a specific gravity of 1012, and was free from albumen. Under the use of the preparations of steel, the ethereal tincture of the muriate of iron, the lactate of iron, &c., the appetite increased; but the paleness, the feeling of weakness, and the other symptoms of anæmia, remained unchanged, notwithstanding a full diet. The patient left the Hospital, and did not return until the 16th of February, 1854. The anæmia had now increased to hydræmia; anasarca and ascites had supervened; there were from six to ten thin pale stools daily; a tender induration, which, however, was not very accurately circumscribed, could be felt upon palpation in the cæcal region; urine 1007, pale and without albumen; blood drawn off by a cupping-glass showed no increase of the white corpuscles. Vegetable and mineral astringents, nux vomica, &c.,

were administered in vain to check the diarrhœa. On the eve of the 25th, loss of consciousness set in suddenly, the spe became stammering, the features distorted, and the eyes fi and staring; pupils dilated; pulse slow and thready; the respirat slow and feeble. On the 26th he died.

Autopsy.

The contents of the cranial cavity presented nothing abnorm The bronchi were empty, and the lungs very œdematous; the he appeared unchanged in its muscular tissue and valvular apparat About four pounds of clear serum were found in the abdomi cavity. The mucous membrane of the stomach and of the sm intestine, as far as the ileo-cæcal valve, was pale. The cæcum o its greater extent was firmly adherent to the fossa iliaca, and mucous membrane was partially converted into an ulcerated su face of a dirty-grey ragged character; the walls of the cæcu presented a pulpy infiltration about five lines in thickness. T lower portion of the intestinal canal, as also the retro-periton glands remained intact.

The spleen was slightly enlarged, five inches long, and three breadth; its consistence was firm, and its cut surface glistening.

The liver was of normal size; its surface was smooth, and its margi sharp; the secreting cells had an irregular outline, and most of the contained no nucleus, but were filled with fine granules and dro of oil, and some of them also with brown pigment. The gall-bladd contained a small quantity of yellow mucus, and in the gall duc within the liver were aggregated masses of cylindrical epitheliu of a grey colour. The liver contained no sugar, but large quantiti of leucine and tyrosine.

The patient attributed his illness to an obstinate intermittent feve from which he had suffered during three months, 2½ years befo his death. The altered conditions of the spleen and liver, whic must be regarded as constituting the starting-point of the anæmi apparently dated from that period. The spleen, which was scarce enlarged, was in a state of lardaceous degeneration, and the liv was affected with fatty degeneration, such as we find as a final resu of infiltration of the parenchyma with albuminous matter, in co sequence of malarious poison, constitutional syphilis, &c., whic may or may not be accompanied by lardaceous degeneration of t spleen, kidneys, and lymphatic glands. The functions of the liver under these circumstances were in a great measure destroyed, and the results, which the suspension of these functions entails upon the entire system, ensued.

The limited cancerous deposit in the cæcum, which did not make its appearance until the symptoms of anæmia had already existed for two years, might have contributed to expedite the death from exhaustion, but could not be regarded as the cause of the poverty of blood.

In the present state of our knowledge, the diagnosis of fatty infiltration, like that of fatty degeneration, is in many cases uncertain. The disadvantage arising from this, however, in medical practice, so far as it concerns the former condition or the simple fatty liver, is not very important, inasmuch as the affection very seldom comes under treatment. The slighter forms exist without causing any important derangement of the general system; the more advanced forms, such as react injuriously upon the system, are often associated with other dangerous morbid conditions, as for instance, pulmonary tubercle, which make the treatment of the hepatic affection a very secondary matter. The cases, in which the liver affection exists *per se*, are usually the result of improper living, hereditary predisposition, &c.; they may be recognised with some degree of certainty from the etiological agencies which give rise to them, in connection with the diagnostic measures already described, either directly, or by way of exclusion.

IX. *Treatment.*

The treatment of fatty liver, which has for its object the diminution of the fatty contents of the liver, may be regarded in several points of view. First comes the regulation of the diet: fat and amylaceous articles of diet, as well as spirituous liquids of every sort should be avoided; fruits are beneficial, as well as those vegetables which are rich in pectine,* and in compounds of the alkalies with the vegetable acids, and also the lean flesh of young animals; active exercise in the open air is to be enjoined, and in short, a mode of life which shall have the effect of hastening the metamorphosis of matter. Those medicinal agents are best suited for the diminution and removal of the fat deposited in the hepatic parenchyma, from which, so far as our present experience in this matter extends, we may expect an increase in the secretion of bile.[*] In the choice of medicines, we must attend carefully to the condition of the digestive organs, and must avoid everything, by the persistent use of which the functions of these organs might be deranged.

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* These are carrots, turnips, and some pulpy fruits, such as apples, pears, red currants, &c.—TRANSL.

In the slighter forms of the affection, it is sufficient, along with a regulated mode of life, to administer the bitter vegetable substances abounding in alkalies, such as the extract. saponar.,[†] the extract card. benz.,[‡] extract of taraxacum, extract of chelidonium,[§] &c., either alone or in combination with alkaline carbonates, or the compounds of the alkalies with vegetable acids; we may also recommend rhubarb, or aloes, when there is great torpidity of the bowels.

In the more advanced forms, it is usually necessary to have recourse to the waters of Karlsbad, Marienbad, Homburg, or Kissingen,[||] of which that should be selected which is most suited to the individual case, to the general symptoms, and to the degree of derangement of the functions of the gastric and intestinal mucous membrane. When there is a marked tendency to diarrhœa, it is best to avoid all the springs just mentioned, and to employ in their place the springs of Eger[¶] or Ems.[**] In anæmic individuals, the easily-absorbed preparations of steel, such as the lactate and carbonate of iron, or, better still, small quantities of the waters of Spa, or Schwalbach,[††] are not unfrequently necessary. When there is persistent exhausting diarrhœa, we must have recourse to the vegetable and mineral astringents.

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[*] Few of the observations bearing on this point, which we possess, can be depended upon. It is only in recent times, that the experiments which have been made upon animals by means of biliary fistulæ, have furnished some reliable data. The information, however, which has been derived from this source, is still far from sufficient to meet the demands of treatment.

[†] Prepared from the root of the *Saponaria officinalis*, or soapwort.—Transl.

[‡] Extract prepared from the leaves of the *Carduus benedictus*, a species of thistle.—Transl.

[§] *Chelidonium majus*, or celandine.—Transl.

[||] See note, page 125.

[¶] The Springs of Eger, in Bohemia, are saline chalybeates, and contain a considerable amount of free carbonic acid. The solid constituents consist of the sulphate and carbonate of soda, chloride of sodium, and carbonate of iron.—Transl.

[**] See note, page 125.

[††] See note, page 121.

The treatment of fatty degeneration of the liver is mainly preventive and symptomatic; the object here is to avert as soon as possible the circumstances which may lead to the liver becoming infiltrated with albuminous matter, and to remove this substance before the nutrition of the cells becomes impaired. Constitutional syphilis, rickets, and other diseases of the bones, the marsh poison, &c., are to be combated by an appropriate treatment; the infiltration of the liver is to be removed by means of iodide of potassium, iodide of iron, the alkalies, and the waters of Karlsbad and other similar springs. It is seldom that we succeed in accomplishing this last indication. I have seen the volume of the liver reduced by such means, but at the same time the symptoms of degeneration often supervened in a threatening manner. When this is the case, the prognosis is unfavourable, and deobstruent medicines are then prejudicial; the treatment must now be confined to the use of the weaker preparations of steel, a bland nutritious diet, and those remedies which are best adapted to regulate the digestion of the stomach and bowels, such as bitter vegetable substances, &c.

CHAPTER VIII.

THE PIGMENT-LIVER.—MELANÆMIC LIVER.—ALTERATIONS IN THE LIVER RESULTING FROM INTERMITTENT FEVERS.

1. *Historical Account.*

THERE is an old tradition, that black substances are formed in the spleen and in the blood of the portal vein, and may give rise to disease; the so-called black bile constituted an important element of the earliest theories of humoral pathology. Galen thought that this substance accumulated in the spleen, as a product of the formation of bile, and that from that locality it gave rise to obstructions of the vessels, enlargements (*Anschoppungen*) of the abdominal viscera, and dangerous nervous symptoms. This theory, with a few intermissions (*Van Helmont, Sylvius*), and with some slight modifications, continued to be resorted to for the explanation of abdominal affections generally, and of diseases of the portal system in particular. The mode of origin, and the pathological importance of atra-biliary matter, we find discussed in detail by Boerhaave* and Van Swieten.† These observers were of opinion, that owing to morbid influences of a varied nature, the fluid elements of the blood were diminished, and the solid constituents condensed into a black, fatty, earthy substance.‡ This substance, it was believed, in virtue of its peculiar nature, accumulated in the portal vein, stagnated here for a long period, assumed acrid and corrosive characters, deranged the functions of the abdominal viscera, and ultimately was converted into black bile.‖ This black bile was then set in motion by certain exciting causes, and, penetrating into the liver, the heart, the lungs, and the brain, produced most serious derangements everywhere:—at one time fever, when the substance had become putrid; at another time, when it led to mechanical obstruction of the cerebral vessels, dangerous symptoms of another character, such as convulsions, paralysis, delirium, &c.*

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* Aphorismi de cognosc. et curand. morb.

† Comment. ad Boerhaav. aphorism. Tom. III., p. 461, *et seq.*

‡ § 1092.

‖ § 1098, 1102.

The theories in question received a considerable expansion from Kämpf,† who added to the atra-biliary matters a series of other substances, which he regarded as proceeding from the blood-plasma.

It was not until the end of last century that Reil, who in this, as in so many other questions, anticipated the age in which he lived, showed how the doctrine of black bile was opposed to physiological observations.‡ After him, Heusinger § attributed the morbid condition under consideration to a deposit of black pigment, and, along with Puchelt, accounted for it by an exaggerated venous condition of the blood. The majority of physicians, however, continued, as before, to believe in the dreaded pathogenetic potency of the black bile. The marsh fevers of the tropics, and the diseases which in 1826 devastated the littoral districts of the northwest of Germany and the Netherlands, were regarded as atra-biliary fevers. Even in the year 1829, Vogel described the *atra bilis* after the fashion of the ancients in the Berlin Encyclopædia.

Scientific medicine had scarcely removed this traditional remnant of a Hippocratic humoral pathology, when it became necessary, from an unprejudiced observation of facts, to relapse into the old doctrine. Diseases became known in which black matter produced by decomposition of the blood, became developed in the spleen, which black matter passed into the portal vein, and at one time obstructed the hepatic vessels, at another, passed through these, and, entering the general circulation, filled up the capillaries of the brain and other organs,—morbid processes, in fact, which gave rise to symptoms similar to those which were described by the ancients.

It is only in the most recent times that accurate observations have been made of this lesion, although isolated instances are found recorded by the older physicians. Long ago, Lancisi ‖ found the liver of an individual who had died of bilious fever, of a blackish tinge; and Stoll ¶ described a dark pigmentary deposit in the brain and liver of a female who had succumbed after several attacks of fever. The statements made by Bailly[*] in his "Pathological Anatomy of Intermittent Fever," are more copious and to the point:—"Le foie tout entier était noirâtre, semblait composé de sang noir," &c.—"la couleur du cerveau beaucoup plus foncée." Simultaneously, Billard described the same change in the brains of three patients who died of acute cerebral disease;[†] and observations of a similar nature are found in Montfauçon's "*Histoire médicale des marais*" (pp. 306-322).

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* § 1104.

† *Op.* ant. *cit.* p. 14.

‡ Reil. Memorab. clinic. med. pract., Fasc. III., p. 54.

§ Untersuchungen über anomale Kohlen-und Pigmentbildung im menschlichen Körper. Eisenach, 1823.

‖ De noxiis paludum effluviis.

¶ Ratio medendi, Tom. I., p. 196.

Black pigmentary deposits were repeatedly observed in the spleen, liver, and brain, during the fever epidemic which, in 1826, raged along the coast of the North Sea.[‡]

Richard Bright, in his "*Reports of Medical Cases,*" published in London in 1831 (Plates XVII., XIX., and Chap. CI.), gives a masterly representation of a brain, the cortical substance of which was of a dark colour, like black-lead.[§] This was taken from a man who died of cerebral paralysis, after an attack of fever. Physicians, who have had an opportunity of observing intermittent and remittent fevers in hot climates, have frequently described a black tinging of the spleen and liver; particularly Annesley,[‖] Haspel,[¶] Stewardson,[**] and others.

All these observations remained without further results, because the mode of origin and distribution of the pigment were not accurately traced. Not until the year 1837 did Meckel[††] ascertain that the dark colour of the organs depended upon an accumulation of pigment in the blood; two years later, Virchow found numerous pigment cells in the blood and in the enlarged spleen of a man who became dropsical after a persistent intermittent fever.[‡‡] Heschl[§§] and Planer[‖‖] have recorded numerous observations of a similar nature.

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[*] Traité anat. patholog. des fièvres intermittentes. Paris, 1825, p. 181, &c.

[†] Archiv génér., 1825.

[‡] Popken, Historia epidem. malignæ Jeveræ observ. Bremæ, 1827. Fricke, Bericht über seine Reise nach Holland im Jahre, 1826.

[§] "It was almost of the colour of black-lead."

[‖] *Op. cit.*, vol. II., p. 482:—"Liver of very dark colour."

[¶] Maladies de l'Algérie, I., 335; II., 318.

[**] The American Journal, April, 1841, p. 42.

[††] Zeitschr. für Psychiatrie von Damerow, 1847; also, Deutsche Klinik, 1850.

[‡‡] His "Archiv für Patholog. Anatomie," 1849 and 1853.

[§§] Zeitschrift der Gesellschaft de Aerzte in Wien. 1850.

[‖‖] The same for 1854.

II. *Anatomical Description of Pigment-Liver, and of the co-existing conditions of the Spleen, Brain, Kidneys, &c.*

In individuals who die from the effects of marsh poison, under symptoms of severe intermittent, remittent, or continued fevers, we frequently find peculiar changes of the liver associated with functional derangements of the organ, and of the parts pertaining to the portal system. The liver presents a steel-grey, or blackish, or not unfrequently a chocolate colour; brown insulated figures are observed upon a dark ground. This change of colour is produced by pigment-matter which is accumulated in the vascular apparatus of the gland. In fine sections of the hardened tissue, accumulations of pigment may be observed in the capillary network of the portal and hepatic veins, as also in the larger branches of these vessels: these deposits are either uniformly distributed, or limited, for the most part, to certain regions. Sometimes the brownish-coloured lobules appear surrounded by black margins, owing to the interlobular veins being filled with coloured particles (Fig. 31); in general, however, the pigment-matter is more uniformly distributed, extending from the circumference of the lobules halfway to their centre, or penetrating as far as the commencements of the hepatic veins, or, still farther, into the vena cava.

In most cases, the arterial system is implicated as well as the venous, the branches of the hepatic artery containing large quantities of black colouring-matter.* The hepatic cells remain exempt; in no case have I observed any deposit of pigment in them, such as is described by Virchow.† They are either normal in their characters, or filled with brown bile; they are often infiltrated with oil, and occasionally, but only after a long continuance of the disease, they contain colloid or lardaceous matter. In the acute cases, the size of the entire organ appears either normal or enlarged; the gland is swollen from congestion, and here and there, also, it is infiltrated and softened by extravasations of blood; at a later period, it diminishes in volume, and there frequently ensues a true atrophy, provided that the gland does not become infiltrated with colloid matter,—an occurrence which I have only met with in rare instances.

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* Not only in malarious fevers, but very frequently, also under other circumstances, as in cirrhosis, cancer, and echinococci of the liver, the hepatic artery contains black pigment, apparently owing to interruptions in the capillary circulation of this vessel.

† After extravasations of blood into the hepatic parenchyma, red, brown, and black deposits of pigment in the cells are not unfrequently observed; in one case of cirrhosis I met with extensive masses of this nature.

FIG. 31.

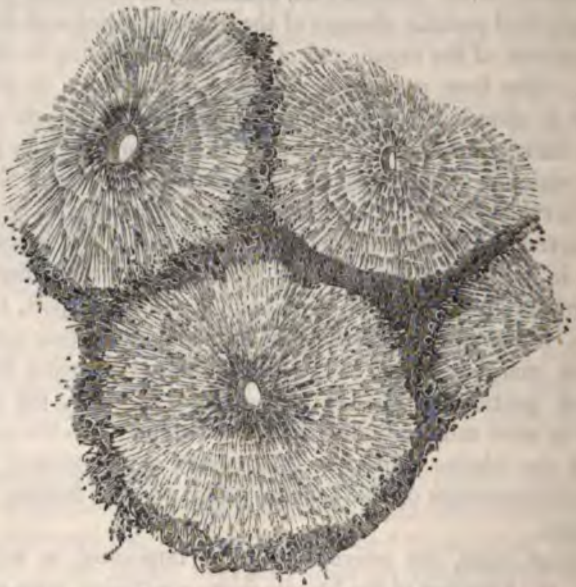

FIG. 31. Fine section of a pigment-liver, previously dried and treated with a solution of potash; magnified 90 diameters. The pigment is seen to be deposited, for the most part, in the interlobular veins, forming a black zone surrounding each lobule, and to have penetrated but very slightly into the interior of the lobules. In most cases, the pigment is distributed more uniformly than is here represented.

Copied from Atlas, Plate IX., Fig. 4.

Along with these abnormal conditions of the liver, we constantly find similar appearances in the spleen. This organ is likewise dark-brown, or sometimes bluish-black; at one time it presents a uniform colour, and at another, is speckled; in its parenchyma large quantities of the same pigment can be detected as is present in the liver. At the same time, the size and consistence of the organ become altered; in the acute cases of the disease, it is usually soft, congested, and considerably enlarged; in the less severe cases, there is usually only a slight alteration in the volume of the organ, unless, as seldom happens, it undergoes lardaceous degeneration; in this case, its volume and consistence are considerably increased. Analogous changes are observed in the lymphatic glands.

The liver and the spleen are the parts in which the black pigment is most constantly observed under the circumstances just mentioned. Very frequently, however, there are other organs implicated as well, the pigment, when it passes in large quantity into the circulation, being carried to them by the current of the blood.

A large quantity has usually been found in the capillaries of the lungs; in old individuals, however, in whom pigmentary deposits of another nature take place in these organs, it is difficult to distinguish between the two.

This tinging with pigment is much more easily detected in the brain, in which considerable collections are sometimes recognised by the dark-colouring of the cortical substance. This assumes a chocolate or black-lead-like hue, whilst the white matter remains unchanged; it is only when the pigmentary deposit is excessive, that we see the white matter present a grey appearance, and the fine vessels in it resembling brown streaks. Under such circumstances, microscopic examination has shown the capillaries to be filled with black granules and scales (*Schollen*), which at one time are uniformly distributed, but at another are aggregated in groups. Frequently we observe along with the pigment scales a pale hyaline coagulum, which when viewed with a light of a strongly refracting power, may be seen to fill up the calibre of individual capillaries.

The kidneys frequently participate in the pigmentary deposit to a remarkable extent. When this is the case, grey spots make their appearance in the cortical substance; and in a few cases, we may observe dark lines in the pyramids following the course of the blood-vessels and uriniferous tubes. Under the microscope we can detect the pigment-matter in the capillaries of the cortical substance, and particularly in the Malpighian bodies; now and then we find isolated little fragments in the uriniferous tubes.

The remaining organs and tissues, such as the external integument, the mucous membranes, the muscular tissue, &c., by no means remain exempt, as may at once be seen by a simple inspection of their grey tint; but the accumulation is seldom so considerable in these parts as in the organs previously mentioned, and it appears to be of subordinate importance, owing to the comparatively slight physiological value of these structures. In the typical forms of the disease we find, as a general rule, pigment deposited everywhere where the blood penetrates, and independently of the abdominal glandular organs, we find it in greatest abundance in those parts of the body where the capillaries are most narrow, and consequently where the pigment flakes are most easily arrested.

The pigment which is found in the organs and tissues, is brought to them by the blood;* it exists in abundance in this fluid, and particularly in that portion of it contained in the portal vein, where we can most easily study its properties, colour, and chemical relations. The usual form in which the pigment makes its appearance is that of small, rounded, or angular granules, which at one time are sharply defined, and at another are surrounded by a brownish or pale margin. These granules are occasionally isolated, but most frequently several of them are connected together in groups, by a pale substance soluble in acetic acid and in caustic alkalies. In form they are rounded or elongated, sausage-shaped or irregularly branched. They have no defined membranous outline; the hyaline connecting-substance which presents the characters of fibrinous matter, forms sometimes a broad, and sometimes a narrow rim, without any sharply-defined outline.

True pigment cells are observed along with the granules and granular masses, though in somewhat smaller quantity. These partly resemble in form and size the colourless corpuscles of the blood, and partly consist of larger spindle-, or club-shaped cells with rounded nuclei, and sharply-defined walls, like the cells which we are wont to find, along with free granules, in the spleen. These cells contain a greater or smaller number of black granules. Planer was unable to convince himself of the presence of these pigment cells, which Virchow had previously described; I have seldom failed to find them in the blood of the portal vein; in most cases it is easy to avoid confounding these cells with the granular masses, which are bound together by fibrinous material. (Fig. 32.)

Besides the forms just described, larger fragments of pigment are observed, which, for the most part, have an irregular form, and upon pressure, look as if they had been broken off from still larger masses. Sometimes these fragments appear to be cylindrical, and are bounded laterally by two straight parallel lines, whilst their extremities are irregularly broken off; so that they remind one of the appearance of the smaller vessels, of which they appear to be casts. Their size is not unfrequently considerable; I have seen some which were $\frac{1}{100}$ of a line in breadth, and $\frac{1}{20}$ of a line in length. They are usually surrounded by a rim of pale transparent substance of greater or less breadth; sometimes this rim is only visible on one side. (Fig. 32.)

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* In the capillaries of the hepatic artery, and occasionally also in those of the portal vein, the pigment appears to lie in the walls of the vessel.

FIG. 32.

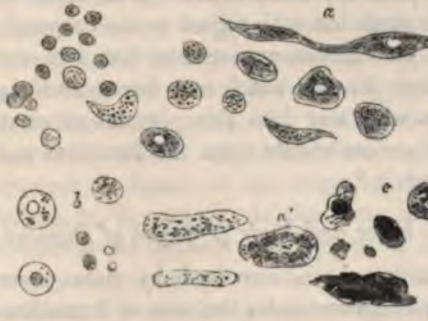

FIG. 32. Pigment-matter from the portal veins.
a, From the trunk of the vessel. The epithelium of the lining membrane containing black pigment, and some of the cells with a distinct rounded or spindle-shaped nucleus; the younger cells were coloured reddish-brown; normal blood corpuscles.
b, Cylindrical scales (*Schollen*) containing pigment.
c, Fragments of black pigment, enveloped in a hyaline substance, from the splenic vein.
a', From the splenic vein.
Copied from Atlas, Plate IX., Fig. 2.

The colour of the pigment is, for the most part, deep black, more rarely it is brown or ochre-coloured, and least frequently of all, reddish-yellow. These different colours represent the various stages in the transformation of the red pigment of the blood into melanotic matter. The gradually advancing metamorphosis, however, manifests itself not merely in the colour, but also in its relations to re-agents. The resistance offered by the different sorts of pigment to the action of acids and caustic alkalies is very variable. The more recent products are rendered pale, and lose their colour with greater or less rapidity; those, on the other hand, of an earlier date, resist the action of these agents for a long time; I have not unfrequently permitted the latter to be acted on for days by caustic soda, without their colour being destroyed.

Along with this pigment, coagula of a hyaline character, and free from colouring-matter, are found in the blood. These resemble in their form the last described variety of pigment flakes; from the fact of their being colourless, they are easily overlooked. Nothing unusual can be detected in the blood corpuscles; sometimes the number of the white corpuscles appears to be increased,—a fact long ago observed by Meckel, but by no means of universal occurrence; in the majority of cases, and especially in those of an acute nature, nothing abnormal can be made out with regard to the corpuscles.

Such are the *post-mortem* appearances found in melanæmic individuals. The first question which now forces itself upon us, is:—Whence, and in what way does this pigment originate?—and the second:—What are its results upon the functions and textures of the individual organs?

III. *Place and Mode of Origin of Pigment.*

This question has been answered by most observers, to the effect that the spleen constitutes the seat of formation of the melanotic matter. There are many grounds for such an opinion, but no proofs exist which can claim this function for the spleen exclusively. Experience shows that the transformation of the red matter of the blood into black pigment may take place everywhere thoughout the vascular system, and also external to it. Although the spleen, from the nature of its structure and circulation appears preeminently adapted for such a purpose, we must keep in view, *à priori*, the possibility of the same blood metamorphoses taking place in other parts of the body, and we cannot arrive at the conclusion that these changes are exclusively confined to the spleen, until observation lends no support to the participation of other parts of the vascular system.

There can be no doubt, however, that the largest portion of the pigment is formed in the spleen, that it passes from this organ into the portal vein, and that part of it remains impacted in the capillaries of the liver, while the rest passes through these capillaries, and is carried into the general circulation.

There are many facts which may be appealed to in favour of such an hypothesis. It is known, that in the spleen of men and animals, and particularly in the spleen of the naked amphibia, cell-like structures are met with, even in the healthy state, which contain blood corpuscles or pigment molecules. It is true, that similar formations are occasionally found in other parts of the body, as in extravasations of blood in the brain, but nowhere so generally as in the spleen. In melanæmia, the accumulation of pigment is not so constant in any organ as in the spleen;—this is a rule almost without exception. Next to the spleen, as regards the frequency and intensity of the deposit of pigment, comes the liver, and after this the remaining organs, the lungs, the brain, the kidneys, &c. Cases are not unfrequent, where only the spleen contains a quantity of pigment, and others, in which the spleen and liver alone are infiltrated with dark colouring-matter, whilst the other organs preserve their normal hue; but I have never observed an accumulation of pigment in the blood of the heart, in the capillaries of the brain, kidneys, &c., without the large glandular organs of the abdomen being implicated. Another argument, in favour of the origin of the pigment of melanæmic patients in the spleen, is the form in which the black pigment is found in the blood. In portions of blood-clot, I have observed the pigment contained in the same elementary structures as are present in the splenic pulp, colourless blood-corpuscles, with a simple or divided nucleus, and spindle- and club-shaped epithelium cells from the cavernous sinus of the spleen. The supposition that other organs are implicated in the formation of pigment, rarely finds any support from examination. The epithelium of the endocardium, and of the lining membrane of the vessels in different regions of the body, does not present any unusual appearance; the accumulation of pigment in the liver, the brain, the kidneys, &c., is always limited to the finest capillaries; extensive deposits which might cause the blood to become overloaded with pigment are nowhere to be found. Only in one case have I been able to ascertain with certainty, that the formation of the black pigment is not exclusively confined to the spleen, but occurs also in the liver. In an individual who died of exhaustion, after a protracted quartan fever, complicated with albuminuria, the enlarged and lardaceous spleen was completely free from pigment, whilst considerable quantities were accumulated in the liver. Not only did this pigment fill the finer capillaries of the portal and hepatic veins, but it also extended into the larger branches of the portal veins; these were blocked up at many places with black, friable coagula, and might be easily traced with a pocket lens, or with the naked eye. Besides the liver, the parenchyma of the kidneys contained much pigment, whilst that of the brain remained free.

Hence, it would seem that the usual source of the formation of pigment is the spleen, and that, along with it, but only in exceptional cases, other organs, and particularly the liver, participate.

It is a more difficult matter to reply to the other question, which has reference to the manner in which this formation of pigment takes place. We shall not solve this problem until we possess a more intimate knowledge of the minute structure of the splenic pulp than we do at present. If we assume, what in the present state of the question appears the more probable view, that the capillaries of the splenic artery pour their blood into a system of large sinuses, whence it passes into the efferent veins, the following explanation of the production of the pigment might be offered. Even in the normal state, the blood which has flowed rapidly through the narrow capillaries, would pass more slowly through the ample channel of the venous sinuses, and not unfrequently would stagnate in some places, so that conglomerate masses of blood corpuscles would be formed, which gradually might be transformed into pigment. In the intense congestions which experience shows to attack the spleen as a consequence of intermittent fever, these stagnations are very great, and would lead to considerable accumulation of pigment. According to this view, the pigment would arise from the stagnation of the blood in the venous sinuses of the spleen; the club- and spindle-shaped pigment cells are the epithelium of the lining membrane of the sinuses infiltrated with the decomposed red matter of the blood, the globular pigment cells are colourless blood corpuscles, infiltrated with molecules of colouring-matter, and the pigment scales (*Schollen*) are the broken-up fragments of the coagulum. Why the formation of pigment is either absent or less marked* in many other congestions of the spleen, in typhus, pyæmia, and simple intermittent fever, why it is, that the metamorphosis of the blood-pigment into melanotic matter, apparently takes place much more rapidly here than at other places, are questions to which at present we cannot give a satisfactory reply. The acid character of the splenic pulp, however, must exercise an important influence over the metamorphosis of the blood-pigment.

Another point of importance for the clinical elucidation of the consequences of this morbid process, would be the tracing of the chemical products of metamorphosis which are formed by the destruction of the blood corpuscles, the morphological residue of which presents itself in the form of pigment. It is scarcely to be imagined, that amid so great a destruction of the elements of blood, products of metamorphosis should not be formed, concerning which the microscope can furnish no information. These are carried along with the pigment into the circulation, and will be taken into consideration in explaining the nervous symptoms, which we know to accompany malignant intermittent fever.*

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* See Observations No. XIII. and XVI.

IV. *The Results produced by the formation of Pigment upon the Structure and Functions of the various organs.*

The effects upon the system of the morbid process taking place in the splenic parenchyma which has just been described, are of a complicated nature, and can only be understood by a close investigation of the individual agents which are concerned in them.

The destruction of large numbers of the blood corpuscles contributes, in the first place, to the production of a state of the blood like that of chlorosis, which usually soon makes its appearance in the course of intermittent fevers. This effect upon the general mass of the blood is aggravated by the interruption of the important blood-forming function of the spleen which takes place at the same time. It is a difficult matter to determine to what extent each of these agencies is calculated to act; the loss of blood corpuscles, however, must be proportioned to their destruction, and consequently to the quantity of pigment which has been formed.† An increase of the colourless corpuscles, as a consequence of the disease of the spleen, is not observed: in most cases the examination of the blood has shown no remarkable deviation in this respect.

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* Boerhaave and Van Swieten (*loc. cit.*, III., p. 496) long ago assumed the existence of a putrefaction of the atra-biliary matter, and attributed the symptoms which were induced by this, partly to the pollution of the blood by the products of putrefaction, and partly to the mechanical obstruction of the capillaries by the black masses. It is obvious that the detection of these products was not to be thought of in the condition of chemical science at that time. Even at the present day, the same difficulty presents itself in explaining the problem before us, because the intermediate substances which arise from the decomposition of the albuminous principles have no characteristic properties, and cannot be detected until the decomposition has arrived at certain ultimate products. Such being the case, negative results cannot decide the question.

We have repeatedly examined the spleen of melanæmic patients, and have found numerous products of metamorphosis which are developed in large quantities in this organ, but have failed to detect any characteristic new substance foreign to the organ.

† The loss in this way may be very considerable. Occasionally the

The pigment originating in the spleen, passes first with the blood of the portal vein into the liver. It is here that the first functional derangements are observed. One portion of the pigment passes through the capillaries unarrested and enters the general circulation; the larger particles remain impacted in the capillaries of the portal vein, and obstruct the circulation of the blood through these vessels. The accumulation of pigment is sometimes seen to take place chiefly at the periphery of the lobules in the interlobular veins; but at other times it extends throughout the entire capillary vascular system, and penetrates into the interior of the lobules as far as the central veins. The derangements of the circulation and their consequences vary according to the quantity of the large pigment scales and cells.

These derangements manifest themselves first in an abnormal secretion of the liver. The bile is usually secreted in large quantity, and we have repeatedly found in it considerable quantities of albumen; leucine can always be detected in the hepatic parenchyma; its saccharine constituents, however, are unchanged.

Extensive capillary stagnation gives rise to obstruction of the circulation of the blood in the roots of the portal vein, which manifests itself in various ways according to its intensity. Sometimes we see exhausting hæmorrhages from the gastro-intestinal mucous membrane coming on in an intermittent manner, or more frequently, profuse diarrhœas, occasionally associated with vomiting, &c.; acute serous effusions into the peritoneal sac, together with bloody suffusions of the serous membrane, constitute another result; while at a later period, chronic atrophy of the liver, with its train of consequences, may make its appearance.

The next organ, in point of frequency to the liver, which undergoes important organic and functional derangements, is the brain. Numerous particles of pigment, which have passed unarrested through the vessels of the liver and of the lungs, accumulate in the narrow capillaries of this organ, and particularly in those of the cortical substance. Even by simple inspection of the shade of colour, we can form an

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organ, which is almost black from the accumulation of blood, is considerably enlarged, diminished in its consistence, and, at some parts, infiltrated with extravasations of blood.

approximative notion of the quantity of colouring-matter which has been deposited, and of the extent of the vascular obstruction. We must not, however, rely entirely upon inspection, for slight accumulations of pigment in the capillaries easily escape notice, particularly when viewed by an unpractised eye, and can only be distinguished with the assistance of the microscope. In addition to the above, it is not at all uncommon for the vessels to become obstructed by a colourless fibrinous-like coagulum, which of course does not affect the shade of colour. The mechanical interruption to the circulation which is produced in this way, not unfrequently gives rise to rupture of the small vessels, and the formation of numerous capillary apoplexies. Meckel long ago made observations of this nature; Planer described eight cases in which small extravasations were scattered through the grey and white substance of the brain. These numerous hæmorrhages have not come under my own observation; but in two cases I have observed extravasation into the meninges.

It has not been proved by direct examinations whether, besides hæmorrhages, other organic lesions of the brain, such as atrophy from interrupted supply of plasma, result from occlusion of the capillaries.

I have seen pigment brains of old date without any remarkable diminution of the cortical substance. Other functional derangements, indicative of material changes in the cortical substance of the brain, have only occurred to me in three cases; these cases, however, I only saw in a cursory manner during a journey in Poland.*

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* One of these was a lady in her fortieth year, who, after an attack of quotidian fever, accompanied by somnolence, suffered from protracted loss of memory. The functions of vegetative life resumed their normal condition, and there were no derangements of motion or sensation present. The headache and giddiness gradually diminished after the removal of the intermittent fever by means of quinine; but the weakness of memory and the inability to find suitable words for objects and ideas were still on the increase two months after the cessation of the ague.

Another case was that of a girl, aged 9, living in the same district, where, according to the evidence of the medical men, intermittent fever, terminating fatally, was at the time very prevalent. This girl, whose mental powers had previously been normal, had undergone several attacks of tertian fever. After a protracted use of preparations of bark, she recovered in her bodily symptoms; but her mental faculties gave way, and a state of complete idiocy, accompanied by a ravenous appetite, supervened.

In these two cases, it is uncertain whether atrophy of the brain had

Along with the structural changes of the brain which have just been described, remarkable aberrations of the functions of the organ are usually observed during life.

These derangements vary in their nature; sometimes they intermit, and cease with each paroxysm of fever, but more frequently they are protracted into the period of the intermission, or are continuous. Even in the last case, however, there are usually unmistakable indications of more or less distinct remissions.

The nature of the cerebral disturbances manifests itself in very various ways; in the slighter cases, they assume the form of headache and giddiness; in the more severe, that of delirium, but more usually of coma, and not unfrequently, there are disturbances of mobility, convulsions, or paralysis. The most constant symptom is dull pain in the head extending over the entire vertex, and almost always associated with attacks of giddiness. This pain was observed in every case in which the loss of consciousness had not arrested all power to communicate subjective sensations; occasionally, the pain was so severe as to cause the patients to utter loud cries. The headache was frequently accompanied by derangements of the senses, noises in the ears, deafness, black vision, and dimness of sight, and occasionally by nausea, a tendency to vomit and vomiting.

Delirium is less frequent; sometimes it is quiet, like that usually met with in typhus; but at other times it is violent, and associated with great excitement and restlessness, so that it is necessary to fasten the patient in bed. By degrees the excitement passes into somnolence and deep coma, which is the most usual form of cerebral disturbance. In several cases giddiness, without any headache, has existed for a long period after the removal of the fever, and this has even been of such intensity that the patients have fallen down suddenly while walking, while at the same time, the anæmia has not been so

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resulted from occlusion of the capillaries, or whether it had been induced by the extensive capillary apoplexies consequent upon this occlusion, or whether the intermittent fever was complicated with other accidental changes in the brain.

It is worthy of notice, that Sydenham (*Opera med. Genev.*, 1736, Tom. I., Sect. 1, Cap. V., p. 60) long ago made mention of mental derangements, which remained after attacks of intermittent fever, and which, under the employment of depleting treatment, soon passed into a state of imbecility. He was surprised that no one had made mention of an occurrence which he himself had so frequently met with.

great as to account for the occurrence. (*Febris vertiginosa Paccinotti.*)

The slight forms of somnolence from which the patient can be roused by loud calling, so as to give rational answers, in most cases after a short time lapse into deep stupor. This stupor sometimes ceases during the intermission, to return with the next paroxsym.

Abnormal conditions of mobility, convulsions and paralyses are much less frequent than derangements of the mental faculties. I have met with convulsions in eight cases; at one time they appeared as slight spasmodic movements of individual muscles of the trunk and extremities, at another time as rapid, rotating, or tossing movements of the extremities and head, and, again, as general convulsions, similar to those of epilepsy, which usually lasted from five to ten minutes, and returned after a shorter or longer interval. Paralysis was only present as an exception. It attacked at one time the muscles concerned in articulation or swallowing, and at another, the extremities; in the latter case, the paralysis was either double; or confined to one side. In one case I have seen the paralysis make its appearance suddenly, and here capillary hæmorrhage was found; in another case, however, in which there was simply an accumulation of pigment, the development of the paralysis was gradual.

That the deposit of pigment in the substance of the brain and the abnormal cerebral symptoms are connected with one another as cause and effect, is a probable supposition, which acquires the more weight from the fact, that in general there is an unmistakable correspondence between the intensity of the two. Hence, the earlier observers, and especially Planer, had no hesitation in attributing the cerebral symptoms to the occlusion of the capillaries with pigment.

I cannot give my unconditional support to this view, however plausible it may at first sight appear, because a close analysis of the observations, and a careful comparison of the anatomical lesions with the symptoms present during life, render the connection between the two, as cause and effect, in many cases, at least, doubtful. Extensive interruptions to the circulation, with capillary apoplexies of the cortical substance of the brain, indicating rupture of the vessels and capillary hæmorrhage, constitute unquestionably a sufficient anatomical basis for the explanation of the cerebral symptoms, but obstruction to the circulation of the blood is by no means always present, even when the colour is dark. The greater part of the pigment passes freely through the capillaries, and may be found in large quantities in the veins. The dark contents of the capillaries are nowhere so distinctly marked as in the white ground of the substance of the brain; hence they always appear more remarkable in this place than in other organs, of which the parenchyma is naturally darker. Even when the circulation of the blood does become arrested here and there, this is not to be regarded as a sufficient cause of functional disturbances, because the numerous branches of the vascular network permit of collateral currents.

On comparing the *post-mortem* appearances with the symptoms during life, we find, on the one hand cases where, in spite of the dark colouring of the brain, no cerebral disturbances have been present, and on the other hand, cerebral disturbances without any deposit of pigment. The last statement I have found to hold good six times, out of 28 cases of *Intermittens cephalica.* The older observers, such as Lancisi, Senac, Bailly, and also Maillot and Haspel, have made similar observations. We cannot, therefore, doubt, that the cerebral disturbances of intermittent fever above described, may occur without melanæmia, and that, consequently, there must be other causes, which can give rise to them. Hitherto, we have been unable to detect these causes. The fact of the morbid symptoms intermitting appears to argue in favour of their being periodically generated in the system, and again disappearing. I have already called attention to the circumstance, that in melanæmia, the extensive decomposition of the blood pours periodically into the circulation, not merely the debris of the morphological disintegration of this fluid, but also the products of its chemical metamorphosis, which elude microscopic examination. A closer study of these metamorphoses may bring us nearer to the source of the cerebral symptoms.

The third organ in which we observe abnormal conditions of structure and functions in melanæmia, is the kidneys. The larger pigment cells and scales which enter these organs along with the arterial blood, not unfrequently become impacted in the capillary coils of the Malpighian bodies, and by altering the pressure of the blood, give rise to derangements in the secretion of urine, which exercise a powerful influence over the further progress of the disease. Albuminuria makes its appearance to an extent which varies with the quantity of pigment found in the kidneys. In the cases where the fever has presented a distinctly intermittent type, and where, as in quartan fever, the intermissions have been of considerable duration, we have observed during each paroxysm a great increase of the albuminous contents of the urine, and during the intermissions a marked diminution, or a complete disappearance of them.

The albuminuria is frequently simple, and then the process may last a long time without leading to intimate structural changes in the kidneys. But in other cases, fibrinous matter passes off in the urine along with the albumen; I have repeatedly observed fibrinous casts, including pigment masses and granules of the same nature as those found in the blood; I have, moreover, met with cases in which bloody urine was excreted. Complete suppression of the urinary secretion is of frequent occurrence. In cases where the secretion of albumen and fibrinous matter has lasted for a long time along with intermittent fever, or after its cessation, and ultimately has led to a fatal termination from exhaustion, alterations of comparative insignificance have been found in the kidneys. Numerous flat, scarred depressions have been observed on the outer surface of the organ, but no distinct granulations. In some cases there has been lardaceous degeneration.

With the exception of the liver, the brain, and the kidneys, in no other organ or tissue can any remarkable change of structure, or derangement of function, be observed. It is true that we can detect pigment throughout the entire capillary vascular system, so far as this is filled with blood, but accumulations of this pigment, and occlusion of the capillary vessels, are only wont to take place to any extent where the smallnesss of the capillaries, or the peculiar nature of their distribution, impedes the free motion of the pigment scales. The supposed production of dyspnœa and œdema of the lungs, from occlusion of the pulmonary capillaries, is not supported by actual results, at least, in the cases which have come under my own observation. I do not believe that the inflammatory swellings which I have observed in isolated cases, such as limited deposits in the parotid and in the muscular tissue of the heart, are the result of extensive occlusion of the vessels. Close examination of the deposits in the muscular tissue of the heart discloses no remarkable accumulations of pigment.

The peculiar colour of the skin which is produced by the quantity of pigment in the blood contained in the vessels of the cutis, is of importance in diagnosis. In the slighter grades of melanæmia it is ash-coloured; in the more intense forms it is dirty greyish-brown, and sometimes deep yellow-brown. In most cases, a few drops of blood, obtained by scarification of the skin, suffices to show numerous particles of pigment, under the microscope.

v. *Pigment-Liver an accompaniment of Malarious Fevers.*

The derangements just described are ushered in and accompanied by a fever, which may assume very various forms and types.

In most of the cases the fever is an intermittent, and usually a quotidian or double tertian,—more rarely a simple tertian, and least frequently a quartan. The fever does not, as a rule, make its appearance in the regular form with distinctly-marked stages, but the intermission is usually incomplete; in most cases, the elevation of temperature and the increased frequency of pulse do not disappear completely, although the repeated attacks of rigors with subsequent aggravation of the symptoms indicate the true type of the fever. After two or three incomplete intermissions, followed by a paroxysm, the fever in most cases becomes continued; patients have been repeatedly brought to the Hospital in this condition as cases of typhus. As a general rule, the intermittent type of fever is less marked, the more prominent are the local, and particularly the cerebral, derangements.

The pulse varies very greatly in frequency. In the most severe forms it does not usually exceed 80 or 90, which is an important circumstance for distinguishing the affection from typhus; in a few cases only has the pulse reached 120 or 140. Sometimes the the pulse abates in frequency upon the supervention of nervous symptoms.

The three stages of the intermittent are seldom very distinctly marked; the rigor stage is often entirely absent, and distinct critical discharges by the skin and urine do not usually occur. Twice I have met with paroxysms of unusual duration (48 and 60 hours). The severe symptoms which usually accompany this form of intermittent, as a rule, make their appearance along with the fever. Sometimes severe brain symptoms, which speedily prove fatal, are developed after only a few hours of vague indisposition, without any distinct febrile symptoms manifesting themselves.

In other cases, a simple intermittent fever has existed for weeks or months, when suddenly a severe fit comes on, which often terminates fatally in an instant. This last form has occurred frequently in relapses of apparently simple tertian or quartan fever.

The cases vary in their progress: there are some which prove fatal in a few hours or days, whilst others are protracted over months; the cases with head-symptoms are almost always acute, the remainder are as frequently chronic. Out of 51 cases which I have observed, there were 24 acute and 27 chronic.

Such are the most important derangements which are observed in the disease under consideration. They seldom all exist together; in most cases one or other of them occupies a prominent place, while the remainder are either absent or developed in a milder form. In this way there arise varieties of disease which exhibit a wide difference in their progress and results. Four forms may be distinguished:—

I. Cases with predominant brain-symptoms.

II. Cases in which the kidneys are pre-eminently implicated.

III. Cases with predominant derangements of the gastro-intestinal tract, and of the appertaining glands, but particularly of the liver.

IV. In a fourth group, we may include those forms in which the local derangements are not very conspicuous, and do not influence essentially the further progress of the disease, but where the anæmia and hydræmia, resulting from affection of the spleen, constitute the most important morbid conditions. The pigment contained in the blood is here of subordinate importance, inasmuch as its quantity and characters do not give rise to extensive lesions of the capillary circulation; it is productive of no injurious consequences, provided we succeed in checking the anæmia.

In order to give some idea of the frequency of the individual derangements, and of their fundamental anatomical lesions, I may annex a short analysis of 51 cases observed here in Breslau. The results are, of course, only of value as regards the epidemic of this place, and must by no means be generalized indiscriminately.

Of the 51 cases, 38 terminated fatally, and 13 in recovery.* Severe brain-symptoms, such as delirium, convulsions, coma, &c., occurred 28 times out of the 51 cases; in 7 of these cases, there was no deposit of pigment in the brain; in 2 cases there was hæmorrhage in the cerebral membranes along with the pigment; and there was 1 case of cysticercus cerebri.

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* Of the cases which ran a favourable course, only those are here included in which pigment could be detected in the blood. These figures do not express the ratio of mortality, because the examination of the blood was often omitted, where no severe symptoms called for it.

Out of the 51 observations, albuminuria was found in 20 cases, in 2 of which there was hæmaturia, and in 5, suppression of urine; albuminuria occurred without any pigment in 4 cases, whereof 2 were instances of lardaceous degeneration of the kidneys; in 5 cases pigment could be detected where there had been no albumen in the urine, but the quantity was scanty.

In 17 of the 51 observations, there was profuse diarrhœa, and 5 of these were cases of dysentery; profuse intestinal hæmorrhage was seen three times. Jaundice was present in 11 cases, but was always slight; bile-pigment also was observed in the serous effusions of the pleural cavities, without any distinct colouring of the skin and urine.

In all the cases which terminated fatally, the liver contained a quantity of pigment; in 10 it appeared enlarged and congested, and in 8 atrophied; in 9 cases the cells contained much oil; lardaceous matter could be detected in 3 cases, but only in small quantity.

Except in one case, pigment was always found in the spleen; three times this organ was lardaceous; and in 30 cases its volume exceeded the usual limits.

In a treatise upon affections of the liver, the cases which come under the first and second groups of melanæmia require only a passing consideration. I annex a few examples, in order to show that the liver is involved at the same time as other parts; and those cases only will be given in detail where the disease of the liver, and the disturbances resulting therefrom, were conspicuous.

I. CASES WITH CEREBRAL DISTURBANCE.

Observation No. XXXII.

Tertian Intermittent Fever of three months' duration, ultimately with Coma during the fit.—Pneumonia, passing on to Consolidation.—Sudden Death.

Pigment-Spleen and-Liver.—Consolidation of the Lungs.—The Brain free from Pigment.

W. Klein, aged 65, locksmith, was admitted in an unconscious condition on the 7th of January, 1855. He had been suffering for three months from tertian fever, which latterly had taken on an irregular type, and, during the fits, was attended by loss of consciousness. On the 8th, he regained the use of his mental faculties. On examination, the spleen was found to be considerably enlarged; on the left side of the thorax there was dulness and bronchial breathing, extending from the middle of the scapula as far as the base; there was no expectoration. Pulse 96. The patient stated that during a frost, eight days before, he had felt a shooting-pain in the left side, and had expectorated saffron-coloured sputa.

The next attack of fever was cut short by bark, in combination with sal-ammoniac.

The bronchial breathing and dulness continued unchanged, and were accompanied by a slight expectoration of grey mucus. The pulse varied in frequency between 80 and 90; the appetite was normal, the bowels regular, and the sleep quiet; neither albuminuria nor œdema was present. There was considerable anæmia.

He was ordered the ammoniated sesquichloride of iron.

On the morning of the 21st, the patient ate his breakfast; and while sitting upon a stool, in order to have his bed made, he became unconscious, and died.

Autopsy, 24 *hours after death.*

Neither the membranes nor the substance of the brain contained much blood; there was nothing abnormal either in the colour or the consistence of the brain. No pigment was found in the capillaries of the cortical substance.

The mucous membrane of the bronchi was pale; the right lung was dry, bloodless, and emphysematous; the left was very firmly adherent to the costal wall; it was diminished in size; its tissue was firm, and not friable; its cut surface was very slightly granular, and of a uniform pale-brown colour; the bronchial tubes were somewhat enlarged, and their lining membrane reddened; the upper lobe contained but little blood.

The heart contained loosely-coagulated dark blood.

The mucous membrane of the stomach and intestinal canal was pale.

The spleen was enlarged by about one-third, and slightly wrinkled; its parenchyma was flabby, tenacious (*zähe*), and of a bluish-black colour.

The liver was of normal size, its colour was blackish-brown. Its consistence was normal.

The bile was thick and yellow.

The cortical portion of the kidneys was atrophied.

The bladder and prostate were normal.

Observation No. XXXIII.

Febrile Gastric Catarrh.—Vertigo.—Convulsions—Coma.—Return of Consciousness.—Parotid Swelling.—Albuminuria.—Death from Exhaustion.

Melanæmia.—Accumulation of Pigment in the Spleen, Liver, the Grey Matter of the Brain, and the Kidneys.

Rosine Hornig, the wife of a day-labourer, aged 61, came to the Hospital on the 22nd of August, 1854, after having suffered for four days from loss of appetite, headache, and giddiness. On examination, there was found to be consolidation of the apices of both lungs; the tongue was coated grey; there was slight distention of the epigastrium, and moderate enlargement of the spleen. The pulse varied between 80 and 90; there had been no rigors.

On the 24th, the patient became restless, complained of severe headache, and was, when in this state, suddenly seized with general convulsions, which, with short intervals, continued for about two hours, and was followed by complete loss of consciousness. On the 25th, the consciousness had not returned. Pulse 84. The urine drawn off with the catheter, contained a considerable quantity of albumen, but no fibrinous coagula; involuntary stools; great restlessness; much sighing and groaning, so that it became necessary to remove the patient to a separate ward.

Here, as we were told, she was tolerably quiet; her consciousness gradually returned, but her memory continued bad, and she only gave slow and unsatisfactory answers. A few days after, an inflammatory exudation made its appearance at the angle of the lower jaw, in the areolar tissue surrounding the parotid. By poulticing, this became soft, burst and discharged a large quantity of putrid pus. The patient then became more and more collapsed, and died from exhaustion, without any return of the convulsions, or loss of consciousness.

Autopsy, Sept. 11th, 20 hours after death.

The bones of the cranium and the membranes of the brain were congested; the cortical portion of the brain appeared of a chocolate colour, and formed a marked contrast to the white substance; this last was of proper consistence and contained the normal amount of blood. The microscope showed that there was a large quantity of pigment included in the capillary vessels.

Grey tubercular matter was infiltrated through the apices of both lungs; lower down, their parenchyma was moderately congested, but free from deposit.

The blood in the heart was coagulated, and of a dark colour. Numerous pigment cells and scales could be detected in it.

The spleen was enlarged by one-half, and was pulpy, soft, and of a dirty greyish-brown colour; numerous brown and black pigment cells and granules, like those in the blood, were found in this organ.

The liver had sharp margins and a smooth surface; its parenchyma was of normal consistence and of a greyish-brown colour; some of its secreting cells contained much oil. The bile was pale and contained no albumen. In the stomach there was the cicatrix of a deep ulcer; its mucous membrane was pale; as was also the mucous membrane of the small and large intestines.

The kidneys were apparently normal; but, on closer examination, a tolerably large quantity of pigment was found in the glomeruli, and uriniferous tubes.

Observation No. XXXIV.

Quotidian Fever.—Enlargement of the Spleen.—Coma.—Death.—Melanæmia.—Accumulation of Pigment in the Spleen, Liver, Kidneys, and cortical portion of the Brain.

Elizabeth Ermler, a tailor's widow, aged 45, came to the Hospital on the 13th September, 1854. For a week before, she had had daily attacks of fever; but previous to this, she had enjoyed perfect health. Her statements, however, seemed scarcely deserving of credit, owing to loss of memory and deafness, which had lasted for eight days. She could not say whether the fever had been ushered in by a rigor-stage. The respiratory and circulating organs were normal; the pulse was 90, small, and soft; tongue dry; abdomen soft and flat; no diarrhœa. The spleen extended about an inch beyond the false ribs; no roseolar eruption. The colour of the skin was pale greyish-yellow, but this tinge was not very striking; the temperature was elevated.

She was ordered muriatic acid with bark.

The state of unconsciousness rapidly increased into complete coma. The countenance became pale, and the pupils small. The temperature continued high; and the pulse varied from 90 to 100. The urine, which came away involuntarily, was not examined. Death took place on Sept. 18.

Autopsy, 20 *hours after death.*

The membranes of the brain were tolerably congested, and the sinuses contained small colourless coagula. The cortical substance of the brain was of a leaden hue, and there was a considerable quantity of pigment granules and scales in its capillaries; the white substance of the brain was of normal consistence. A small quantity of serum was contained in the sac of the arachnoid.

The mucous membrane of the air-passages was pale; dark obsolete tissue was found at the apices of both lungs, accompanied on the right side by enlargements of some of the bronchi, which contained a calcareous pulp; posteriorly and inferiorly, there was œdema.

The heart contained a small quantity of loosely coagulated blood, in which numerous pigment particles could be detected.

The spleen was enlarged by about one-third; it was flabby, and its surface was wrinkled; its tissue was soft and of a greyish-brown colour.

There was a tight-lace mark upon both lobes of the liver; the surface of the organ was smooth, its parenchyma friable and chocolate-coloured; from the lower surface of the left lobe there projected the sac of an echinococcus undergoing disintegration. The hepatic cells were loaded with brown pigment, and contained but little oil.

The mucous membrane of the stomach was tumid, and of a livid hue; in the small and large intestine it presented, at some places, a slaty-grey colour.

The kidneys were apparently normal, and the glomeruli loaded with pigment. A small quantity of slightly albuminous urine was found in the urinary bladder. The genital organs presented nothing abnormal.

OBSERVATION No. XXXV.

Fever of an undecided type.—Delirium.—Coma.—Vomiting.—Death on the fifteenth day.—No albuminuria.

Pigment in the Blood, the Cortical portion of the Brain, the Liver, Kidneys and Spleen.—Spleen slightly enlarged.

Bertha Meissner, aged 38, sempstress, was admitted in an unconscious condition on September 11th, 1854. She had been ill for fourteen days; and after suffering from delirium and great restlessness, she had since the beginning of the last week completely lost her consciousness. The patient was of a pale, dirty-yellow colour; her head and extremities were cool; her eyes were coated over with mucus; the pupils were of medium size, and acted somewhat slowly. The pulse was 84 and small; no enlargement of the spleen could be made out. The abdomen was tender; thin, deep-brown stools were passed involuntarily. The urine was free from albumen.

Notwithstanding the employment of stimulants, the patient became very rapidly collapsed; and 14 hours after her admission she was a corpse. I did not observe any attack of rigors, nor were any noticed by the relatives.

Autopsy, 15 *hours after death.*

The inner surface of the skullcap was covered by a thin layer of bony growths; the dura mater was at some places firmly adherent; the pia mater was anæmic; there was a small quantity of clear serum effused beneath the arachnoid. The cortical substance of the brain was of a dark ash-grey colour; its capillaries were filled with pigment granules. The white substance of the brain was anæmic and of normal consistence; the lateral ventricles were of the usual size.

The air-passages and the lungs presented little that was abnormal; the posterior and inferior portions of the lungs were hypostatic and œdematous; the deposit of pigment in them was considerable.

The heart, as well as the large vessels, was normal. The blood, both in the right and left ventricles, was rich in pigment.

The stomach had a somewhat thickened, slaty-grey mucous membrane, presenting a few ecchymoses upon the prominent folds. The mesenteric glands and the intestinal canal were healthy.

The spleen was slightly enlarged; it lay very far back; its parenchyma was very soft, and dirty greyish-brown; brown and black pigment in the form of scales and cells could be detected in it in abundance.

The liver was of normal size; its surface was smooth and its edges sharp; its parenchyma was soft and of a dirty greyish-brown colour; the secreting cells appeared pale; the capillaries were filled with pigment. The gall-bladder contained a quantity of thin, mucous, pale bile.

The surface of the kidneys was smooth; their parenchyma was shrivelled and anæmic; the glomeruli contained pigment scales. The urinary bladder was quite empty.

The uterus was that of a virgin; and there were small corpora lutea in both ovaries.

OBSERVATION No. XXXVI.

Intermittent Fever with Convulsions and loss of Consciousness.—Type irregular.—No rigor stage.—Recovery by treatment with bark.

Carl Grund, aged 28, was admitted on the 27th of October, 1854. His illness commenced fourteen days before, and was characterised by persistent headache, accompanied by noises in the ears, nausea, and great weakness and delirium; there was no evidence, however, of rigor, or of any of the other symptoms of intermittent fever. On the 27th of October, soon after admission, the patient was seized with an attack of convulsions, accompanied by loss of consciousness, which lasted about half-an-hour and returned once more during the night. On the morning of the 28th, a considerable enlargement of the spleen was detected; pulse 78; great headache, but consciousness clear; the skin secreting actively; the urine throwing down a slight deposit, but containing no albumen; stools normal.

The increased frequency of the pulse and elevation of the temperature appeared at irregular intervals, at one time daily, and at another time every second day, and were accompanied by giddiness and severe headaches, passing into delirium; these symptoms gradually subsided with sweating. They were never ushered in by a rigor. During the intermissions, the patient's condition was but little deranged. On the 8th of November, he was ordered 3 grains of quinine every two hours; under the use of this, the headache, giddiness, and excitement of the vascular system ceased; but on the 16th they returned in a severe and regular tertian form, though still without any rigor stage. Bark was administered for a long time; and under its use the symptoms gradually disappeared.

These attacks left the patient in a very low state, and he required a regulated chalybeate treatment, before he could be dismissed from the Hospital cured, and free from any enlargement of the spleen.

Observation No. XXXVII.

Intermittent Fever of an irregular type.—Two paroxysms of 48 *hours' duration.—Furious Delirium during the paroxysms.—Cure.*

Heinr. K. Gürtler, aged 39, a year before had suffered from intermittent fever for several weeks, and at the end of July was seized with violent pains in the region of the spleen, accompanied by moderate enlargement of the organ. Along with this there was bronchial catarrh, without any fever. He was treated at the Polyclinique with muriate of ammonia, and afterwards with senega.

At noon on June 29th, a moderately severe rigor came on, lasting about an hour, and followed by heat of skin, violent headache and furious delirium. Towards evening, the patient became completely unconscious; pulse 130; the skin dropping with perspiration. Not until noon of July 1st, did the sweating and frequency of pulse abate, and the consciousness return. In the evening, the patient was free from fever.

He was ordered three grains of sulphate of quinine every two hours.

On the afternoon of the 5th, the temperature became elevated; pulse 92 and *bisferiens;* tongue dry; severe headache. This condition lasted until the 7th, when profuse sweating and sudamina made their appearance, the pulse falling to 70.

By the continued use of quinine the patient rapidly recovered, and on the 16th was able to leave the Hospital.

OBSERVATION No. XXXVIII.

Quotidian Intermittent with severe Vertigo.—Febris vertiginosa of Paccinotti.—Cure by Quinine.

Carl Forster, aged 30, since the middle of September, had suffered from a quotidian intermittent, associated with severe vertigo. The patient could scarcely advance two steps without staggering; on attempting to walk across the room, he fell repeatedly, and was obliged to be led. The colour of his skin was greyish-brown, like that of a mulatto; the spleen was enlarged; there was no albumen in the urine. The pulse became more frequent; neither headache, rigors, nor any of the other symptoms of fever were present; the appetite was unimpaired, and the stools normal.

The patient having been treated before admission into Hospital, by cupping and purgatives, without any benefit, recourse was now had to quinine, whereupon the giddiness rapidly diminished, and ere long entirely ceased.

In a few days, the patient was able to be discharged from the Hospital, there being no anæmia to necessitate a longer treatment.

II. CASES WITH PREDOMINANT AFFECTION OF THE KIDNEYS.

OBSERVATION No. XXXIX.

Quotidian Intermittent of four weeks' duration; Diarrhœa; Albuminuria and Hæmaturia; Sudden Stupor; Convulsions; Death.

Accumulation of Pigment in the Spleen, Liver, Kidneys, and Brain.

C. Runschke, aged 50, came to the Clinique on the 3rd August, 1854. For four weeks he had suffered from a quotidian fever, which had latterly become complicated with diarrhœa. His complexion was of an extremely pale greyish-yellow hue; there was no œdema, however, anywhere. The spleen extended an inch beyond the margin of the false ribs; the abdomen was soft and painless, and there was no ascites; the liver was of normal size; there was nothing abnormal in the respiratory organs or in the heart.

The urine was deep reddish-brown, and coagulated upon boiling, or on the addition of nitric acid. The pulse was 80; and there was much complaint of headache.

Was ordered quinine with opium.

On the 4th of August, the patient stated that he had an attack of rigors during the night; but the nurse had not observed it. The pulse was unchanged, 80, small and weak. The headache was considerably increased. One formed stool.

Ordered quinine with elix. acid. Hall.*

In the night, sudden restlessness and loud groaning. Professor Rühle, who was summoned to the patient, found him in a state of deep coma, with irregular, interrupted respirations, and a small frequent pulse; the pupils were not enlarged; the temperature of the head was increased.

At 7 in the morning, he expired; death being preceded by slight convulsions.

Autopsy, 14 *hours after death.*

The pia mater congested; the dura mater thickened and adherent to the inner surface of the skullcap.

After removing the meninges, the grey substance of the brain appeared of a dark chocolate colour, and was defined by a sharp line from the central white matter. This copious deposit of pigment was very marked in the corpus striatum and cerebellum, and also in the pons Varolii. The white substance was intersected by fine blackish lines; besides this, the brain presented nothing abnormal in consistence, or in any other respect.

The air-passages pale; the lungs congested and œdematous.

In the heart there was a moderate quantity of loosely coagulated dark blood, with much pigment in various forms.

Spleen large, soft, and darkly speckled.

The liver normal in size; surface smooth; margins sharp; its parenchyma loaded with greyish-brown pigment; the bile pale and copious.

The mucous membrane of the stomach was of a slaty-grey colour; the lining membrane of the intestine was perfectly normal. The pancreas contained a larger quantity of pigment than is usual in such cases. The kidneys were lobulated, and, on section, presented a homogeneous, brown, glistening surface. There were numerous pigment cells and scales in the glomeruli of the Malpighian capsules.

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* *Elixir acidum Halleri*, or *Mistura acida sulphurica*, is composed of concentrated sulphuric acid (1 part), and rectified spirit of wine (3 parts). Its specific gravity varies from 1005 to 1010. Dose 5 to 20 minims—TRANSL.

The bladder contained a large quantity of bloody urine, and its walls were hypertrophied. There was a slight stricture of the urethra in front of the bulb.

OBSERVATION No. XL.

Continued Fever, with symptoms like those of Typhus.—Coma.—Albuminous and bloody urine containing clots loaded with black pigment.—Right Pneumonia.—Abortion.—Death on the sixteenth day.

Spleen and Liver soft and loaded with pigment.—Occlusion of the tubuli uriniferi by pigment.

Rosalie Hellmann, aged 28, was admitted on August 25th, 1854, after having been treated for fourteen days, by a physician outside the Hospital, for a disease which was considered to be typhus. The woman was perfectly unconscious; her yellowish-grey skin was covered with perspiration; pulse 120, small and weak; respiration frequent, irregular, and stertorous. The thorax was of normal resonance in front, at some places slightly tympanitic; there was dulness at the lower and back part of the right side. Here bronchial breathing was audible; loud râles were to be heard in front; heart's sounds normal. The patient was in the eighth month of pregnancy.

At 10 in the evening, labour-pains came on, and about 2 in the morning a living child was born. The patient's general state did not improve. The dyspnœa and frequency of the pulse increased; the pulse rose to 136. The skin was warm and trickling with perspiration. The urine drawn off by catheter was bloody, and contained a quantity of albumen and fibrinous coagula. Death took place at 1 P.M.

Autopsy, 16 *hours after death.*

The membranes and substance of the brain were somewhat congested; the consistence and colour of the latter were normal. The mucous membrane of the air-passages was injected and covered with frothy mucus. The upper lobes of both lungs were congested and œdematous; the lower portion of the right lung contained no air, was friable and greyish-yellow, and exuded a purulent fluid upon pressure. The margin of the lower lobe of the left lung was collapsed. The heart was flabby and shrivelled; its valves were normal; the blood in the auricles was loosely coagulated, and contained much pigment.

The spleen was considerably enlarged, pultaceous and chocolate-coloured. The blood of the splenic vein contained much pigment.

The liver was dark-brown, without any distinct indication of lobules; its cut surface was smooth, its margins sharp, and its consistence friable. The cells of the organ were very pale; the bile was dark, thick, and viscid.

The mucous membrane of the stomach was relaxed and injected, and covered here and there with ecchymoses. The mesenteric glands were small, and contained no deposit; the mucous membrane of the small intestine was pale, without any abnormal development of its glandular apparatus; the large intestine was intensely injected.

The kidneys were of normal size; their outer surface was smooth, and they contained a moderate amount of blood. The glomeruli of the Malpighian capsules contained numerous brown and black pigment scales.

In the urinary bladder was a small quantity of bloody urine, containing small, pale, cylindrical clots infiltrated with pigment scales.

Observation No. XLI.

Quartan Intermittent Fever.—Intermittent Albuminuria.—Anasarca.—Dysentery.—Rapid cure by means of Quinine and Iron.

Henriette Schadek, aged 27, wife of a day-labourer, six months' gone with child, came to the Hospital on the 8th of November, suffering from a quartan fever of six weeks' duration. There was much œdema of the upper and lower extremities, and also of the face; the urine was scanty and contained a quantity of albumen, and a number of fibrinous coagula, in some of which black pigment was observed. The spleen extended 4 centimètres ($1\frac{3}{5}$ inch) beyond the margin of the false ribs. The thoracic viscera were normal. It was stated, that the anasarca had made its appearance soon after the commencement of the fever.

The quantity of albumen contained in the urine diminished considerably on the 9th; and on the 10th it almost completely disappeared.

On the 11th a severe paroxysm set in, whereupon the albuminuria returned with renewed severity.

On the 13th there were frequent mucous stools mixed with blood, and accompanied by tenesmus. The fever was cut short by quinine; enemata of tannine and opium were administered to counteract the dysentery.

On the 18th the dysentery ceased, and the albuminuria disappeared.

On the 19th recourse was had to the lactate of iron; the quantity of urine became remarkably increased, and the œdema disappeared; so that on the 8th of December the patient was able to be discharged from the Hospital cured.

III. CASES WITH PREDOMINANT IMPLICATION OF THE LIVER AND GASTRO-INTESTINAL CANAL.

The implication of the liver is distinguished in many cases by a tenderness upon pressure over the right hypochondrium, as well as by an increase in the volume of the gland. These signs, however, may be absent even when the terminal ramifications of the portal vein are filled with pigment. We frequently observe a slight jaundiced tint of the skin and conjunctiva, together with brown bile, or allied colouring-matters in the urine; but these symptoms are not constant.

When the passage of the blood of the portal vein through the liver is interrupted to a great extent, we soon perceive the consequences of obstructive congestion upon the gastro-intestinal mucous membrane and the peritoneum; hæmorrhages take place, or increased secretion from the bowels, profuse diarrhœa, and sometimes also acute ascites supervene.

Intestinal hæmorrhages I have observed on three occasions; the bleedings were intermittent and came on each time with the paroxysm of the fever; they were not affected by any treatment directed against the hæmorrhage, but yielded to large doses of quinine.

The first case of this nature which I met with, terminated fatally; because, from the continued character of the fever, the disease was supposed to be typhus. The case was that of a young man, aged 20, in whom severe intestinal hæmorrhage took place after he had been suffering for fourteen days from what his medical attendants called a slight attack of typhus. The patient, when I saw him, had passed several pounds of dark blood, and was much exhausted. His skin was greyish-yellow; pulse 110, scarcely perceptible. There were, first, three attacks of hæmorrhage, each relapse being preceded by an interval of two days; the attacks took place amidst great excitement of the vascular system, and were apparently arrested by styptics, such as alum, and the muriate of iron. The fourth attack, which also came on after an interval of two days, proved fatal.

Soon after this, I saw another patient at the same place, in whose case, likewise, the diagnosis of mild typhus had been made, from the fact that, after four attacks of a quotidian fever, the intermission had become indistinct; in this case also there was profuse intestinal hæmorrhage, which was subject to daily exacerbations, coming on always about the same hour. Styptics were tried in vain. Under the use of quinine, with elix. acid. Hall.,* the hæmorrhage ceased, and the patient became convalescent.

In a third patient with quartan fever, the intestinal hæmorrhage came on every three days, and was accompanied each time by hæmaturia; here, also, quinine sufficed to effect its removal.

Many cases of a similar nature occurred about the same time; I only mention those which I was able to observe most carefully. I have frequently found, upon *post-mortem* examination, dark suffusions upon the serous covering of the bowels and in the mesentery, resulting from the obstructions to the circulation. It could not be ascertained whether or not these cases were accompanied by intestinal hæmorrhage during life.

In other cases, where the obstruction to the circulation in the portal vein appeared to be less intense, there were profuse secretions from the intestinal mucous membrane, and rapid effusions of serum into the abdominal cavity. Diarrhœa occurred chiefly in individuals who were employed in the waterworks during the inundation of 1854; not unfrequently it passed into dysentery. I am, however, unable to state whether in these cases the interruption to the portal circulation was the only cause, or whether there were any additional causes in operation. Spontaneous catarrh of the bowels, unaccompanied by intermittent fever, was at the time rarely met with.

In cases where the effects of the accumulation of pigment in the portal vein were less marked during the persistence of the intermittent fever, more remote consequences might be observed at a later period. After the obliteration of a portion of the capillaries, the cells of the parenchyma in their neighbourhood disappeared, and there arose a chronic atrophy of the liver, such as has been described in Chap. VI., Observations XXIV. and XXV., pp. 258-9.*

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* See note, p. 343.

OBSERVATION No. XLII.

Persistent and oft-recurring Intermittent Fever, ultimately of a quartan type; Albuminuria of a high grade; fibrinous casts, with pigment in urine.—Œdema.—Rapidly-supervening ascites.—Tapping after the inefficacious employment of steel and purgatives.—Return of the Fever.—Paroxysm of two days' duration.—Death from exhaustion.

Pigment in the Spleen.—Occlusion of the hepatic capillaries.—Atrophy of the Liver.—Accumulation of Pigment in the Kidneys.—Consecutive Pneumonia.

Doroth. Schirmer, aged 38, a labourer's wife, was in the clinical ward from the 17th May to the 31st July. She had suffered repeatedly from intermittent fever of various types, and, during the last winter, from an almost uninterrupted quartan fever. At the time of her admission, the form of the fever was double quartan. For fourteen days, the patient had observed œdema of the feet, and a remarkable diminution in the secretion of urine; the urine was turbid, greyish-yellow, and contained an enormous number of fibrinous casts, which were partly covered by black pigment granules and cells. By boiling, the urine was converted into a solid coagulum. The region of the kidneys was tender upon pressure. The spleen extended about 4 centimètres (1½ inch) beyond the margin of the false ribs. The blood drawn off by means of a cupping-glass (care being taken to avoid the admixture of any foreign matter), contained brownish and black pigment masses, in the form of scales (*Schollen*) and cells.

After taking a drachm of quinine, the attacks of fever ceased. The characters of the urine, however, did not alter, and it continued scanty; there was repeated vomiting of greenish mucous fluid, without any headache or darkening of the countenance. Was ordered lemon-juice. The quantity of urine diminished somewhat, the vomiting ceased, and the appetite improved. Was ordered lactate of iron. The œdema of the feet diminished; but ascites became rapidly developed to such a degree as to interfere with respiration. Colocynth, gamboge, and similar purgatives were of little avail against the dropsy; the energetic employment of these remedies only gave rise to derangements of digestion, vomiting, &c., so that it did not appear advisable to persist in them.

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* Haspel (*Malad. de l'Algérie*, T. I., p. 335) appears to have previously observed a case of this nature in an individual who had suffered from obstinate diarrhœa, with ascites, after an attack of tertian fever, and whom it had been necessary to tap repeatedly; he found the liver small, not easily lacerable, and blackish internally.

On the 10th of July, twelve pounds of clear serum were drawn off by tapping. The volume of the liver, which could now be determined with greater accuracy, was slightly diminished.

On the afternoon of July 12th, there was an attack of fever,—rigors, heat, and sweating; the fever did not return on the 13th.

On the night of the 14th, after the usual interval, notwithstanding the employment of quinine, the paroxysm again made its appearance; the pulse continued very frequent, rose to 140, and was scarcely perceptible, without our being able to detect any cerebral disturbance, dyspnœa, or physical changes in the heart or pericardium. The pulse continued frequent from the evening of the 14th to the morning of the 17th, when it suddenly sank to 88. No fresh attack of fever took place; the pulse remained to the last varying between 80 and 90; but the ascites rapidly increased, and the œdema of the feet spread to a remarkable extent. The patient became rapidly prostrated; and, after a protracted agony, death took place on the 31st, without any cerebral symptoms.

Autopsy, August 1*st,* 12 *hours after death.*

No remarkable alteration was observed in the skullcap, meninges, or substance of the brain, either in their amount of blood, colour, or consistence. The mucous membrane of the air-passages was pale; the left lung was œdematous superiorly; inferiorly, it was infiltrated, but only to a slight extent, with soft exudation; the pleura, at this place, was covered with a thin layer of grey flaky exudation. The right lung was hypostatic posteriorly, and emphysematous along its anterior margin. The pericardium contained about three ounces of clear serum; the muscular tissue and valvular apparatus of the heart were normal. The blood in the right auricle was firmly coagulated.

The œsophagus was pale. The mucous membrane of the stomach was ecchymosed at some places, and, around the pylorus, was of a slaty-grey hue. The lining-membrane of the bowel was pale superiorly, and, in the large intestine, was œdematous, and at some places the larger vessels were injected. The fæces were yellow. The pancreas and mesenteric glands were normal.

The spleen was not enlarged, but flabby and shrivelled; its capsule was thickened; and its parenchyma was tenacious (*zähe*), and of a bluish-grey colour.

The left lobe of the liver had a broad atrophied rim; white traces of obliterated vessels could be seen in its serous covering. The parenchyma had a smooth surface on section, was somewhat friable, and of a greyish-brown colour. The entire volume of the organ was diminished. The gall-bladder was found to be adherent to the stomach and colon, and was greatly distended with a green, mucous, somewhat albuminous bile.

On microscopic examination, the usual forms of black pigment could be detected in the spleen, in the blood of the portal vein, and in the capillaries of the liver.

The kidneys were of normal size; their surface was smooth; their capsule firmly adherent; and their cortical substance greyish-yellow, soft, and friable. The microscope showed the existence of pigment in the glomeruli, as also in the vessels of the cortical substance, and occasionally, also, in the interior of the uriniferous tubes.

The mucous membrane of the bladder was ecchymosed; the urine was scanty and albuminous.

The genital organs were not altered to any great extent; there was a cyst the size of a hazel-nut in the right ovary.

OBSERVATION No. XLIII.

Intermittent Fever of a Tertian and Quotidian Type, lasting for seven weeks.—Intestinal Catarrh.—Hydræmia.—Anasarca.—Improvement under preparations of steel.—Relapse.—Rapid increase of the Dropsy.—Unconsciousness.—Death.

Pigment in the Spleen and Liver, without any implication of the Brain or Kidneys.

Franz Krocker, aged 45, a worker on the roads, came to the Hospital on the 1st of November, 1854. For seven weeks, he had suffered from an intermittent fever, at one time of a tertian, and at another, of a quotidian type; and, four weeks before, anasarca had made its appearance. The spleen was found to be considerably enlarged, extending about three inches beyond the margin of the false ribs; the heart and lungs were normal; there was no albumen in the urine; the bowels were relaxed and the motions thin.

After the arrest of the fever by means of quinine, the patient's condition improved; under the use of nutritious diet, wine, and the preparations of iron, the anasarca receded and the diarrhœa abated until the 8th of November, when the temperature became elevated, the pulse rose to from 96 to 100, and dyspnœa, with sibilus and rhonchus in the air-passages, and acute headache, made their appearance. At the same time, a copious effusion of fluid was detected in the abdominal cavity, and, soon afterwards, in the pleural sacs likewise. Although this change in the patient's condition did not in any way resemble a fever paroxysm, yet considering the uncertain character presented by the malignant forms of fever, recourse was had to quinine. The patient, however, rapidly collapsed; his colour became earthy; somnolence set in, and on the 14th, he died.

Autopsy, 24 hours after death.

The body was greatly swollen from dropsy. The meninges and substance of the brain contained the normal amount of blood. There was no alteration in the colour or consistence of the brain, and no pigment in the capillaries of the cortical substance.

The mucous membrane of the air-passages was slightly reddened, and covered with grey mucus. Several pounds of serum were found in each pleural cavity. The lungs were anæmic, and of a dirty-grey colour, with emphysematous margins superiorly; inferiorly, they were compressed and non-crepitant.

The pericardium contained a pound of clear serum; in the heart, there was loosely coagulated blood, in which pigment was found in tolerable quantity. The muscular tissue and valvular apparatus of the heart were normal.

The peritoneal sac contained many pounds of clear serum. The stomach and intestinal canal were pale, but in other respects healthy.

The spleen was very large; its capsule was tensely distended; its parenchyma was soft and of a dark greyish-brown colour.

The liver was shrivelled up at its margins; the serous membrane along the anterior rim of the left, as well as of the right, lobe, was opaque from recent exudation, and covered with pale-red newly-formed vessels. The hepatic tissue was of a dark greyish-brown colour, and of soft consistence. Here, as well as in the spleen, there were large masses of pigment.

Bile, thin, pale, and albuminous.

Kidneys anæmic.

Urinary bladder and prostate normal.

Observation No. XLIV.

Slight Dysentery. — Albuminuria. — Death from exhaustion. — No cerebral disturbance.

Accumulation of Pigment in the Spleen, Liver, Brain, Kidneys, and Pancreas.

Josepha Weiss, aged 54, wife of a day-labourer, came into the Hospital, on August 29th, 1854.

This woman was in a very low state, emaciated and anæmic; and for three weeks, had been suffering from diarrhœa, accompanied by the passage of bloody mucus and great tenesmus. She referred this diarrhœa to a cold; she had not noticed any attacks of intermittent fever. On admission, the sphincter ani was so relaxed, that bloody mucous stools constantly flowed away from it; the urinary bladder was paralysed; the dark urine drawn off by catheter contained a large quantity of albumen.

A transient improvement was obtained under the use of a suitable nutritious diet, wine, and enemata of decoction of calumba and tannine. Soon, however, the dysenteric affection increased. The exhaustion made rapid progress; the patient became more and more indifferent, slept much, but always gave perfectly intelligible answers. After a protracted agony, death took place on the 9th of September.

Autopsy, 26 hours after death.

The meninges were moderately congested; there was slight œdema of the arachnoid. The cortical substance of the brain was of a slaty-grey colour, and sharply defined from the anæmic white matter.

The consistence was normal. The capillaries of the cortical substance were filled with granules and scales of pigment.

The lungs were dry and anæmic. There was much pigment in the blood contained in the heart.

The spleen was enlarged by about one-third, of a pulpy consistence, and dirty-grey colour, and loaded with black and brown pigment.

The liver was of normal size, soft, and chocolate-coloured, and its secreting cells were pale. The bile was scanty and dark, and contained no albumen. The capillaries of the liver were filled with pigment at the circumference of the lobules, and to a less extent near their centre. The mucous membrane of the stomach was of a slaty-grey hue; the lining membrane of the small intestine was pale. The mucous membrane of the large intestine, from the ileo-colic valve as far as the sphincter ani, was covered with a grey puriform fluid; at many places it was abraded and marked by hæmorrhagic effusions, but it was free from any great amount of exudation, or from losses of substance of a deeper nature.

The pancreas was deeply tinged with pigment. The kidneys were anæmic and shrivelled (*welk*); the glomeruli, as well as the uriniferous tubes, contained a moderate amount of pigment.

There was much turbid urine in the urinary bladder, which, upon boiling, deposited albumen, but contained no casts of tubes. The genital organs were normal.

Observation No. XLV.

Abdominal Typhus.—Repeatedly recurrent Intermittent Fever.—Dysentery.—Exhaustion.—Death.

Spleen and Liver loaded with pigment.—The latter organ atrophied.—Dysenteric ulcerations of the large Intestine.

E. Hahn, a female, aged 59, passed through a slight attack of typhus in September, after which, she was seized with an oft-recurring intermittent fever. In the course of this, and without any other apparent cause, the digestion being tolerably good, and there being no diarrhœa or albuminuria, great anæmia came on within a short space of time: the skin assumed an eminently white appearance, and became dry and desquamating; extensive anasarca set in.

In November, several paroxysms of a quartan type occurred, which were soon removed by means of quinine, but which left behind a diarrhœa, gradually assuming all the characters of dysentery. Muriate of iron, nitrate of silver, with opium and other remedies, were employed to check this, without effect; the œdema rapidly increased; the patient passed into a state of collapse, and died from exhaustion on December 9th, death not being preceded by any loss of consciousness.

Autopsy, 12 *hours after death.*

The brain and its membranes, as well as the organs of respiration and of circulation, did not exhibit any important change, with the exception of paleness and an appearance of anæmia.

The spleen was of the normal size; its tissue was greyish-brown, and of moderate consistence. Its capsule was much wrinkled.

The liver was small; its outer surface was smooth, and its margins sharp; its parenchyma was moderately consistent and bluish-brown. The lobules were surrounded by dark rings. There was a small quantity of dark bile in the gall-bladder. The stomach and pancreas were normal.

The sigmoid flexure was drawn towards the right, and fixed there by bands of false membrane. The serous membrane of the rectum was opaque; its mucous membrane was very tumid, and covered with thick greenish-yellow masses of exudation; scattered over it were numerous patches of ulceration. These morbid changes extended, with a gradually diminishing intensity, as far as the ileo-colic valve. The mucous membrane of the ileum was pale, and presented some grey typhus cicatrices.

The kidneys were rather small, at some places granular, but otherwise normal.

The right ovary contained a simple cyst, the size of one's fist.

VI. *Etiology.*

Considering the great frequency of intermittent fevers, the cases in which there is a marked development of pigment are comparatively rare; hence, in such cases other agencies, of which we possess no accurate information, must co-operate with the usual causes of intermittent fever. In the present defective state of our knowledge as to the nature of infectious diseases (*Infectionskrankheiten*), it cannot be determined whether a particular quality, or an unusual intensity of miasm is necessary for this purpose. The epidemic, to which the cases that I have described, belonged, made its appearance after an inundation in Silesia, resulting from an overflow of the Oder, in 1854. Since then, cases of this nature have very seldom been met with there, although the ordinary forms of intermittent fever are never absent.

VII. *Diagnosis.*

A perfectly accurate diagnosis can only be made by direct examination of the blood: a few drops carefully collected, so as to avoid contamination with any foreign matter, are sufficient to determine the presence or absence of large quantities of pigment. To the practised eye, the peculiar grey, ash-coloured, or greyish-yellow colour of the skin furnishes an obvious means of diagnosis. The supervention of severe cerebral symptoms with the co-existence of albuminuria or hæmaturia, or rapid collapse, is less to be depended on. More reliance is to be placed upon the occurrence of such cases in an epidemic form; this must especially guide us, when severe brain symptoms, intestinal hæmorrhages, suppression of urine, &c., are suddenly developed in the course of a fever of an indistinct type, without any other cause sufficient to account for these derangements. The periodic increase of such symptoms, the comparatively slow pulse, the increased volume of the spleen and liver, the colour of the skin, &c., may supply further data for diagnosis. In some cases, the favourable effects of quinine may of themselves confirm the diagnosis.

VIII. *The Prognosis.*

The prognosis always remains a matter of doubt. The mere cessation of the fever does not entitle us to give a favourable prognosis, because sudden and unexpected relapses, speedily terminating in death, not unfrequently supervene. The cachexia and anæmia, resulting from the changes which the spleen and liver usually undergo, are ominous symptoms. The albuminuria, when it comes on periodically, and has not lasted a long time, yields readily to quinine; when of longer duration, it is often treated without any benefit. Coma and convulsions are in general of bad omen; yet such cases are not unfrequently cured by appropriate and well-timed remedies.

IX. *Treatment.*

The cure of the intermittent fever is the first thing to be done, and the necessity for this is the more urgent, the more severe the symptoms are during the paroxysm; every fresh paroxysm endangers life or increases the sum-total of the derangements which threaten to put an end to it. In such cases, so soon as we are sure of our diagnosis, large doses of quinine are sufficient, and the remedy is best given diluted with acids, in which form it is most easily and rapidly absorbed. The presence of counter indications must not be allowed to occasion delay, unless they are of a very important nature, such as great congestion of the brain.

In the slighter forms, with predominant abdominal derangements, great gastro-enteric catarrh, jaundice, congestive swelling of the liver, &c., it is judicious treatment to counteract these disturbances by suitable remedies, before having recourse to the quinine. This medicine must not be discontinued too early, because relapses are very apt to occur, and are particularly dangerous.

After the removal of the fever, the second indication is the treatment of the remaining local derangements of the spleen, liver, kidneys, brain, &c. Simple enlargements of the spleen usually yield to the use of quinine, and the easily absorbed preparations of steel, such as the hydrochlorate, the lactate, or the citrate of iron. It is a more difficult matter to effect the removal of the colloid infiltration of the organ, which is met with every now and then; for this purpose we must have recourse to the preparations of iodine, particularly the iodide of iron, and the mineral waters containing iodine and bromine, which are to be employed with more or less caution, according to the condition of the blood.

The congestion of the liver usually disappears spontaneously after the cessation of the fever, or even before this; when it remains stationary for a long period, we may employ rhubarb, the extractum saponariæ,* with the neutral salts, the ammoniated sesquichloride of iron, with extract of aloes. The same treatment is applicable in cases of enlargement of the liver, dependent upon biliary obstruction arising from catarrh of the duodenum and hepatic ducts.

A much greater danger, as regards the progress of convalescence, consists in the derangements of the nutrition of the liver, which are apt to result from the capillaries of the organ becoming loaded with pigment, and from the antecedent congestion conjoined with the alteration in the composition of the blood, produced by the marsh poison. What we have most to dread, is a gradually progressing atrophy of the gland, resulting from the destruction of numerous capillaries, the consequences of which we have already become acquainted with in Chapter VI. I know of no treatment, by means of which such atrophy can be averted. We have already considered how the consequences of this atrophy, the gastric catarrh, the exhausting diarrhœa and the ascites are to be treated. The intermittent hæmorrhages and the acute ascites which succeed extensive obstruction of the capillaries, are best met by a speedy arrest of the intermittent fever; not until after this, can we have recourse to astringents and styptics.

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* See note, page 312.

The fatty and the colloid infiltrations of the liver, which occur in other cases, are to be treated according to the general principles laid down in subsequent chapters.

The third organ to be considered in the treatment is the kidneys; they become diseased, in such a manner as to excite apprehension, sometimes at an early date, at other times, not until a later period. Albuminuria and hæmaturia, which accompany the fever paroxysm, and remit and intermit along with it, yield best to quinine, and disappear for the most part, so soon as the fever ceases. When the abnormal condition of the secretion continues after the cessation of the fever, tonic and astringent remedies are suitable, such as the extract of cinchona, dissolved in some aromatic water, tannic or gallic acid, the preparations of iron, &c. A similar treatment, alternated with derivation to the skin and bowels, by means of warm baths on the one hand, and purgatives on the other (these last, however, only to be employed when the mucous membrane of the bowel is unaffected), is adapted to chronic albuminuria and the dropsy, which, in most cases, are developed, as the consequence of the cachexia remaining after the fever. The result in these cases depends chiefly on whether the affections of the spleen and kidneys are accompanied by still more important lesions of the liver, and of the mucous membrane of the stomach and bowels. When these parts are involved, it is seldom that we succeed in arresting the dropsy. When there is obstinate albuminuria, and at the same time a solid enlargement of the spleen, we have reason to believe that there is colloid infiltration of the kidneys, and we must employ the iodide of iron, so long as the condition of the digestive organs and the hydræmia will tolerate it; this is one of the most dangerous forms of the affection.

Cerebral disturbances during the febrile attack, demand a special treatment, when there is great plethora, or when there is a threatening of cerebral paralysis; in the former case, abstractions of blood, with cold applications, are to be employed; in the latter, diffusible stimulants, ether, musk, ammonia, &c. We must not, under these circumstances, neglect the use of quinine. Headache, giddiness, and other derangements, which remain after the removal of the intermittent fever, yield best to the protracted use of this alkaloid.

The altered composition of the blood, the anæmia and the hydræmia, in almost all cases, necessitate a tonic after-treatment, consisting in an easily digested animal diet along with steel. The effects of this mode of treatment soon become apparent, when there are no important local affections in the liver, bowels, or kidneys, to interfere with assimilation, or to keep up abnormal secretions. When this is the case, all our endeavours to effect an improvement often prove fruitless.

CHAPTER IX.

HYPERÆMIA OF THE LIVER, AND ITS CONSEQUENCES.

THE liver, in virtue of its structure and the arrangement of its vessels, is more subject than any other organ to abnormal distributions of its blood. The current of blood, which under the pressure of the heart flows but slowly through the enormous and much ramified vascular network of this gland,—seeing that this pressure has already had to overcome one set of capillaries in the roots of the portal vein,—is acted on by agencies of a peculiar nature, as a result of which its rapidity and tension are liable to many variations.

Besides the heart's action, the organs in which the roots of the portal vein originate, influence the circulation in the liver. During digestion, when the abundant secretion of the mucous membrane of the stomach and bowels causes an active flow of blood to these parts, and when, at the same time, large quantities of fluid are absorbed, the blood flows more actively through the portal vein to the liver than is the case at other times.* Similar consequences may result from the irregular contractions of the splenic vessels, which are amply provided with muscular fibres.

The hepatic circulation is still more affected by the action of the diaphragm and abdominal muscles, by means of which the abdominal cavity is narrowed, the portal vein compressed, and its contents carried towards the constantly open vessels of the liver.† The contractions of the muscular tissue of the stomach and intestines produce a like effect upon the delicate roots of this vein, although in a more limited degree.

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* According to Cl. Bernard, in order to protect the liver from too great a pressure of blood during the period of digestion, special communications exist between the portal vein and vena cava, which carry the blood directly into the vena cava, without passing through the portal capillaries.

† The earlier physicians had peculiar notions as to the circulation of blood in the portal vein, upon which they grounded their theories of obstructions and enlargements *(Anschoppungen)* of the liver. Boerhaave believed that the blood in the portal vein circulated independently of the heart. "Sanguis venæ portarum amittit omnem a corde acceptum impetum (*Prælect. academ.* Ed. Haller, vol. III., p. 115). The blood, he thought, was propelled through the liver by means of the muscular

A third important agency affecting the circulation of the liver, is the varying opposition made to the escape of blood by the hepatic veins. This diminishes during each inspiration, and increases with the expiration. During the inspiration, the blood is drawn towards the heart from the open hepatic vessels, whilst at the same time the portal vein and the liver are compressed by the descent of the diaphragm, and their vascular contents propelled further towards the heart. This movement is favoured by the contraction of the greatly developed muscular tissue of the hepatic veins.

Besides these influences, which for the most part are due to the position of the liver and its vessels, and which are very variable in the extent of their operation, the hepatic circulation is influenced by other agencies, which are less accurately known. Among these may be mentioned the contractility of the walls of the vena porta, as well as of those of the hepatic vein and artery, which appears to be chiefly dependent upon innervation, and which, in rare cases, is liable to modifications from an altered state of their nutrition. When treating of chronic atrophy of the liver, we ascertained that dilatations of the portal vein may result from interruptions to the nutrition of the vascular walls; other forms of derangement arising from a similar cause will afterwards be brought forward. We know little that is positive as to the influence exerted by the nerves over the circulation of blood in the various divisions of the portal vein, and in the hepatic artery. According to the experiments and observations of Cl. Bernard, injuries of certain localities of the medulla oblongata (*Diabetic puncture*), electric irritation of the proximal extremity of the divided vagus nerve, contusions of the head, poisoning with curari, &c., may give rise to hyperæmic swelling of the liver; I have myself observed a remarkable degree of congestion of this organ after division of the splanchnic nerves, and removal of

tissue, with which the portal vein is furnished in the capsule of Glisson (*ibid.* p. 115 to 118), as well as by the respiratory movements of the diaphragm and abdominal muscles (*ibid.* p. 183). Stahl likewise denied the influence of the heart, and thought that the blood of the portal vein was propelled by the respiratory movements, and by a peculiar tonic motive power, having its seat in the spleen, intestines, mesentery, &c. Even at the present day, these views are brought to bear upon the doctrine of abdominal plethora.

the greater part of the cœliac ganglion. As might have been expected, the circulation of the blood in this organ is modified to a greater extent than we are aware of, by nervous influences. These still require investigation, because we are by no means entitled, without further investigations, to apply to the portal vein the results of experiments which have been made on other portions of the vascular system.*

From what has just been said, it will be understood, that in a system of vessels, subject to such manifold modifying agencies, various derangements may occur, the causes of which are at one time obvious, but at another time less intelligible. We confine ourselves in the first place to the simpler forms, the etiology of which is more easily investigated, and shall afterwards pass on to the consideration of the more complicated varieties.

A. *Hyperæmia of the Liver, arising from obstruction to the circulation of the Blood. (Stauungshyperämie.)*

I. *Pathology.*

In valvular diseases of the heart, and especially in such as give rise to accumulations of blood in the venæ cavæ at an early period, such as constriction of the left auriculo-ventricular opening, incompetence of the mitral, and still more of the tricuspid valves, and further in affections of the lungs, which greatly impede the circulation through the pulmonary artery, such as emphysema, extensive induration or atalectasis, in constriction of the thoracic cavity from angular curvature, in great pleuritic effusions, &c., hyperæmias of the liver are met with as a general rule. Under such circumstances, a powerful agency in the circulation of the blood in the liver, viz.: the suction power exercised during inspiration, is either more or less powerful than it ought to be; either the blood in the vena cava and hepatic veins is subjected to a higher degree of pressure, which interferes with the evacuation of the capillaries of the portal vein, or the current of blood regurgitates with each systole of the heart into the hepatic veins, as may be made the subject of direct observation in the case of the veins of the neck.

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* Kölliker and Virchow observed in the body of an executed criminal, that no contraction resulted from galvanic irritation of the trunk of the portal vein, and only a slight contraction in the superior mesenteric vein. In experiments made by myself and Reichert on living dogs, a distinct, although slight, contraction was observed to take place in the splenic and mesenteric veins; in the trunk of the portal vein this was indistinct; no contraction could be observed in the hepatic veins and vena cava inferior, in which, from their very muscular character, the effects might have been expected to have been more remarkable.

When this is the case, the branches of the hepatic vein in the liver continue permanently distended, and gradually enlarge, while their walls become hypertrophied; the stagnation is propagated to the portal vein, and to the organs in which this vessel takes its origin; and there results a series of derangements in the functions and nutrition of the liver and of the organs comprised in the portal system.

The liver enlarges in every direction without any important alteration in its form; its capsule becomes tightly distended, and the consistence of its parenchyma firmer.* On section, the organ presents a nutmeg-like appearance, which can also be seen through the capsule, and which varies in character according to the degree of the congestion. Usually there may be seen rounded, simple, or branched, dark brownish-red patches, surrounded by portions of tissue of a pale-brown colour. The dark spots correspond to the situations of the hepatic veins, and their form depends upon the direction in which the section of the vessel passes.† The light-coloured portions of the parenchyma exhibit upon closer inspection, pale delicate ramifications corresponding to branches of the portal vein.

When the congestion attains a more advanced stage, the dark parts become incorporated together, and enclose in an annular manner portions of the pale tissue which corresponds to the branches of the portal vein; the forms of these pale parts then appear to be, for the most part, rounded or oval, sometimes they are elongated, or divided in a dichotomous manner, and more rarely they resemble a leaf.

In the highest grade of hepatic hyperæmia, the organ exhibits over extensive portions a uniformly dark-red colour, in the midst of which the reddish-black outlines of the greatly distended branches of the hepatic vein can be made out.‡

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* Here and there, the parenchyma is doughy and œdematous, serum can be squeezed out of it, and it retains impressions made upon it with the finger.

† Even in the normal state, the blood is retained here in considerable quantity, and indications of such a nutmeg-appearance are not of very great importance, especially in cases where death has proceeded from disease of the lungs. For other causes of this appearance of the liver, see page 295, note †.

‡ Kiernan (*Transactions of the Royal Society for* 1833) has correctly described this variation in the degree of hyperæmia of the liver, but the

The obstruction in the hepatic veins necessarily interferes with the circulation of the blood throughout the entire capillary system of the liver, and through this, reacts upon the portal vein, as far as its primary roots. The secretion of the liver does not appear to suffer any important change: I have not been able to convince myself that it is either increased or diminished; in a few cases, the bile contains albumen. The mucous membrane of the bile ducts are often seen to become swollen, and to throw out an increased secretion, and in this way the ducts themselves may become partially obstructed, and occasionally, also, a stoppage to the flow of bile, with slight jaundice may be observed. The nutrition of the hepatic cells remains at first unaffected, but after a time, we observe in those which are situated nearest the commencements of the hepatic veins, an abundant deposit of fat, such as in other cases is only met with in the cells next the branches of the portal vein.

From the persistence of this obstruction to the circulation of the blood, there is gradually developed a peculiar atrophy of the hepatic parenchyma, which formerly was frequently confounded with cirrhosis. The gland which hitherto has been swollen from the quantity of blood which it has contained, begins to diminish in size, and at the same time its surface, and afterwards its parenchyma, acquire a finely-granular structure. The granulations arise from the circumstance that the central veins of the lobules, and the capillaries opening into them, or in other words, the roots of the hepatic veins become enlarged under the powerful pressure of the obstructed blood, and thus lead to a disappearance of the hepatic cells enclosed in their meshes. The cells situated in the middle of the lobules become atrophied, and in their place appears a soft vascular tissue, consisting of enlarged capillaries and newly-formed areolar tissue, whilst the cells in the periphery of the lobules nearest the branches of the portal vein remain unaffected.* The farther this atrophy extends,

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numerous delineations of these varieties given by him are far from natural.

* These appearances can be traced best in the injected liver. A magnified representation of such an injected liver is given in the Atlas (Pl. XII., Fig. 4); different coloured injections had first been thrown into the hepatic and portal veins. The central veins are seen to be surrounded by capillaries considerably larger than those met with under normal circumstances; the meshes containing the cells are greatly narrowed, so that at some places they are found to be smaller than the surrounding vascular tubes. As we approach the portal veins, the capillaries

the more does the entire volume of the liver diminish, and the more distinct do the granulations become.

Through the hepatic veins the obstruction is propagated to the portal vein, and to the structures in which the roots of this last vessel take their origin. The vessels of the mucous membrane of the stomach and intestine, of the spleen, pancreas, &c., become distended with venous blood; and, as a consequence of this, their nutrition and secreting powers are deranged. These results manifest themselves first and most distinctly in the mucous membrane of the stomach and bowels; this assumes a more or less dark-red colour, its tissue becomes softened, infiltrated with serous fluid, and consequently swollen; while not unfrequently, extravasations of blood take place, which give rise sometimes to a deposit of black pigment, and sometimes to erosions and ulcerations. The secretion is usually increased in quantity and altered in quality, being thinner and more watery, or it often continues scanty. At the same time, watery effusions take place from the serous surfaces of the abdominal cavity. The spleen increases in volume, but only for a time, and never to a remarkable degree; at a later period, it becomes more solid and firm, and is reduced to its normal dimensions.* In many cases, we find old extravasations of blood between the folds of the mesentery, the mesenteric glands of a livid tinge, with congestive swelling and serous infiltration of the pancreas. In consequence of the increased pressure on the part of the blood of the portal vein, the absorption of the liquefied ingesta, so essential for nutrition, is greatly impeded.

It is worthy of notice, that, although the pressure must be uniformly distributed from the trunk of the portal vein over its radical branches, still the results of the obstruction are by no means uniform in the various parts from which these branches come. Of the gastro-intestinal mucous membrane, it is only some portions which are always seriously diseased, whilst the remainder appear but little changed. Disease of the mucous membrane of the stomach is the

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become smaller, while the meshes are larger, and contain cells of normal character.

* In thirteen cases of organic disease of the heart, in males, the average weight of the spleen was 0.23 kilogrammes (8.1 oz. avoird.); its relative weight to that of the body was as 1 to 276; of nine cases, in females, the absolute weight was 0.17 kilogr. (6 oz. avoird.), and the relative weight as 1 to 282; in thirteen cases, of females, in which there was also extensive dropsy, the absolute weight was 0.14 kilogr. (5 oz. avoird.), and the relative as 1 to 464. See pages 24 and 25.

most constant, and the most marked; out of 20 cases, this membrane was of a dark-red colour in 12, and in 7 it was covered with ecchymoses and erosions; in 4 cases only was the membrane relaxed and of a pale colour. The small intestine is quite different from the stomach in this respect; as a general rule, the lining membrane of this portion of bowel is pale and scarcely altered; in 5 out of 20 cases, only, was the ileum moderately congested, and never to such a degree as the stomach. The large intestine, and particularly the cæcum and sigmoid flexure, have been observed to be congested somewhat oftener, or in 7 out of 20 cases; this congestion has at one time been uniformly distributed over the greater extent of the mucous membrane; at other times, it has been limited more to the folds of the bowel, and to the circumference of the solitary glands; in no case did its intensity equal that of the injection of the stomach.

These inequalities in the distribution of the congestion appear to be dependent upon the differences presented by the individual portions of the intestinal canal, as regards their vascularity, and the size and position of their vessels; besides these causes, there are other circumstances, which in many cases must co-operate, such as the irritation of ingesta or medicines, and also the variations in the degree of *post-mortem* congestion, produced by the accumulation of gas in isolated portions of the bowel.

Independently of the portal system, we find the derangements just described almost always accompanied by albuminuria, which sometimes is persistent, and at other times is only observed from time to time. This morbid condition is of importance as regards the affection of the liver, inasmuch, as all the derangements referrible to the portal vein, including the affection of the liver itself, are less prominently developed, the greater the loss of albumen by the urine, and the more marked the anæmic condition, which is gradually induced by this drain upon the system, as well as by the interference with digestion and intestinal absorption.

II. *Symptoms.*

The several stages in the development of obstructive hyperæmia of the liver, may usually be traced with ease at the bedside of the patient. The enlargement of the gland first makes itself known by a feeling of tightness and heaviness in the right hypochondrium, usually associated with more or less severe symptoms of gastric catarrh, and not unfrequently with slight jaundice. Percussion and palpation at once indicate an increase in the volume of the organ, the extent of which varies with the degree of obstruction to the circulation; during an attack of violent dyspnœa and cyanosis, the dulness soon increases to the extent of several centimètres. The surface of the organ at first feels smooth and tense; at a later period, it is uneven and granular. After a long continuance of the disease, the volume of the organ gradually diminishes; and this diminution is more rapid, the more complete is the obstruction to the circulation, and the earlier the date at which a marked degree of anæmia shows itself.

Besides the local changes in the liver itself, we observe the effects of the obstruction to the portal circulation, manifesting themselves chiefly by derangements* of digestion, pains and tension in the epigastrium, nausea, &c., and occasionally, also, by enlargement of the hæmorrhoidal veins. The bowels, as a rule, are confined; in rare cases, a transient diarrhœa makes its appearance (only in 4 out of 20 cases). The scanty, highly-coloured urine, almost always contains small quantities of albumen, and sometimes also of bile-pigment; the latter of these complications is in most cases only temporary, and is associated with a slight jaundiced tinge of the conjunctiva and skin.

These derangements of the abdominal organs are accompanied by the symptoms of the cardiac or pulmonary disease, which constitutes the primary cause of the obstruction to the circulation, and which, sooner or later, leads to death from pulmonary œdema, apoplexy, general dropsy, &c.

III. *Treatment.*

In the treatment of the hepatic affection under consideration, we must keep in view, that it constitutes only a link in the long series of pathological processes, which are originated and kept up by one common cause, and that it depends upon the nature of this cause, whether the results produced are temporary or permanent.

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* Not unfrequently we find, upon *post-mortem* examination, all the signs of chronic catarrh of the stomach, tumefaction, lividity, a thick layer of mucus, &c., in cases where no indications of any derangement of digestion had existed during life.

In most cases, palliative treatment is all that is possible, inasmuch as the fundamental organic lesions cannot be removed. The main point to be attended to is to reduce the congestion of the portal system, and to moderate the injurious consequences which this produces upon digestion and nutrition. A careful fulfilment of these indications contributes greatly to the relief of the complaints which are necessarily associated with cardiac diseases, and to ward off the dangers to which they give rise. Considerable swelling of the liver, accompanied by pain in the right hypochondrium, is most easily reduced by mild saline purgatives, the action of which may be kept up for a longer period by infusion of rhubarb; at the same time, we may apply cupping-glasses over the hepatic region, and leeches to the anus. Where the obstruction to the circulation is not of too threatening a character, we may often succeed in procuring permanent relief by the careful employment of the Ragoczy Spring of Kissingen, or of the Mill Springs of Karlsbad. I have had under my care a patient suffering from constriction of the left auriculo-ventricular opening, who for four years contrived to pass a tolerable winter by using the cooler springs of Karlsbad; and I have repeatedly obtained similar results by the careful employment of the waters of Marienbad and Kissingen.* In cases where the disease of the valves or of the muscular tissue of the heart is far advanced, the mineral waters can no longer be borne.

On the appearance of dropsy we must avoid saline medicines, and substitute for them the bitter vegetable substances, such as rhubarb, aloes, and similar preparations, to which must be added, in the event of tympanites, aromatics, æthereal oils, and small quantities of ether. When there is great tenderness of the stomach, we must apply a few leeches, or confine ourselves to the employment of the watery tincture of rhubarb with cherry-laurel water, extract of belladonna and similar remedies. Gastric hæmorrhage, which, however, is seldom profuse, and also ulceration of the stomach, call for the employment of ice and astringents.

We must beware of inflicting a lasting injury upon the digestive organs, by persisting in the use of digitalis.

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* See note, pages 58 and 125.

Observation No. XLVI.

Constriction of the left auriculo-ventricular opening of the Heart.—Incompetence of the tricuspid valves.—Marked venous pulse.—Hæmoptysis. — Albuminuria. — Obstructive hyperæmia of the Liver, Stomach, and large intestine.

Carl Scholz, a journeyman baker, aged 38, was admitted into Hospital on December 19th, 1854, and died on the 19th February, 1855. Five years before, the patient had suffered from an attack of acute articular rheumatism, which had been followed by cardiac palpitations, dyspnœa, and bloody expectoration.

On admission, there was marked dyspnœa, extensive râles, and finely-frothy sputa tinged with blood. The impulse of the heart was felt at its normal site, and over the apex there could be heard a systolic and diastolic murmur; the systolic sound was heard still louder over the ensiform process of the sternum; both disappeared in an upward direction; the second sound of the pulmonary artery was unusually loud. The cardiac dulness was increased in width. The countenance, and particularly the lips, were of a cyanotic hue. The veins in the neck were greatly distended, and exhibited systolic pulsation in a marked degree. The pulse in the radial artery was 80 and small. The liver extended about 1½ inch beyond the margin of the ribs, the hepatic dulness in the mammary line amounting to 11 centimètres (4⅓ inches); its surface felt uneven and communicated a sensation of pulsation to the hand placed over it. The tongue was clean; the appetite slightly impaired; and the bowels confined. The urine was scanty, dark, and slightly albuminous.

Was ordered infusion of digitalis with 2 grains of extract of aloes, daily.

The dyspnœa and abdominal tension were relieved, but only for a time. Œdema of the feet and ascites gradually became developed, and there were repeated attacks of hæmorrhagic œdema of the lungs (*blutiges Lungenœdem*), which had to be removed by digitalis and benzoic acid. Up to the beginning of February, the patient continued tolerably comfortable, his appetite kept up, and his sleep was but little disturbed; but from this date, without any alteration in the auscultatory signs above described, the dyspœna, cyanosis, and dropsy daily increased, and were accompanied by complete prostration of the digestive powers, until on the 19th the patient had an attack of asphyxia, which terminated in death.

Autopsy, 17 *hours after death.*

The cerebral membranes were congested and thickened; the substance of the brain was normal. In the neck was observed a remarkable enlargement of the internal jugular vein, which presented a bulbous projection above the clavicle, the size of a walnut; the external jugular vein was likewise enlarged.

The mucous membrane of the air-passages was congested, and at some places suffused with blood; both lungs were crepitant throughout, much congested, but not œdematous. The pericardium was opaque, white and thickened; over the right ventricle, which at its base measured 3¼ inches in breadth, was a large fibrous spot (*Sehnenfleck*); the tricuspid valve was thickened and opaque, its margins were rounded; the chordæ tendineæ, as well as the flaps of the valves, were adherent to one another; in consequence of this the auriculo-ventricular opening was much enlarged, and its valvular apparatus incompetent. The endocardium of the enlarged auricle had an opaque white appearance, the foramen ovale was one line in diameter: the valves of the pulmonary artery were normal. The walls of the left ventricle were unaltered; the bicuspid (mitral) valve was connected to a rigid ring, and at its base were rough calcareous deposits; the coronary veins were much enlarged; the aorta was normal.

The mucous membrane of the stomach was intensely congested, bluish-black, and at some places covered with grey flakes; the lining membrane of the jejunum and ileum was pale; the cæcum and colon were at some places congested and ecchymosed. The mesenteric glands and veins were unaltered.

The spleen was of normal size; its capsule was thickened; and its parenchyma reddish-brown, firm, and congested.

The liver was large, its capsule opaque, and its surface unevenly granular; its parenchyma was very congested; its colour appeared at some places of a uniform dark-red, with black specks corresponding to the hepatic veins; here and there spots could be distinguished, partly white and partly of a greenish-yellow colour, surrounded by dark congested spaces. The vena cava descendens and the hepatic veins were enormously enlarged, and their walls were thickened. This enlarged condition extended, as was shown by injection, as far as the capillaries of the lobules; the hepatic cells were partly atrophied, and partly filled with oil globules; in the greenish-yellow spaces the cells were loaded with bile-pigment. The gall-bladder contained a scanty, thick, dark secretion.

The cortical substance of the kidneys presented a few isolated superficial scars.

Observation No. XLVII.

Constriction of the Left Auriculo-ventricular opening.—Repeated Attacks of œdema of the Lungs.—Albuminuria.—Congestion of the Liver from Obstructed Circulation.—Hæmorrhagic erosions of the Stomach.—Intestinal mucous membrane, pale and tumid.

Veronica Gräser, a joiner's wife, aged 29, was admitted on October 31st, 1854, and died on the 6th of December following. She had already, three years before, been treated in the Hospital for œdema of the lungs, which became developed soon after delivery, in consequence of constriction of the left auriculo-ventricular opening of the heart. Even then the same physical signs could be made out, as on the patient's last admission. A diastolic murmur, associated with *frémissment*, could be heard loudest over the apex of the heart, which was felt to beat at its normal place; this was accompanied by a remarkable exaggeration of the second sound to the left of the sternum in the second intercostal space; the cardiac dulness was increased in its transverse diameter, and extended beyond the right margin of the sternum. The pulse was 90, small and irregular. There was very extensive bronchial catarrh, a moderate amount of effusion in the right pleural sac and in the abdominal cavity, and œdema of the feet. The renal secretion was scanty; and there was a small quantity of albumen in the urine. The liver extended about 2½ inches beyond the margin of the ribs; its dulness in the mammary line amounted to 14 centimètres (5½ inches); the surface of the gland was smooth, its margin sharp, and its consistence firm. There was no appetite, although the tongue was clean; the bowels were somewhat confined, and the stools of a dark-brown colour.

Was ordered infusion of digitalis leaves with tincture of rhubarb and acetate of potash.

The respiration became freer, and there was an increased secretion from the kidneys and the bowels; the œdema of the feet disappeared; the hepatic dulness diminished by about 3½ centimètres (1.378 inch).

Up to the 18th of November, the patient continued to improve under the use of a weak infusion of rhubarb; the digestive powers rallied; the bowels were moved regularly; and the dyspnœa was less violent. On the 18th, the pulse became very irregular; the dyspnœa again increased; there were finely-frothy sputa; the volume of the liver attained its former size. Under the use of the ethereal tincture of digitalis, the rhythm of the heart's action certainly improved; but the nausea, which soon supervened, and the complete loss of appetite, rendered it necessary to exchange this preparation for mild bitter remedies. The urine was always scanty, and its albuminous contents inconsiderable; the ascites and œdema of the feet rapidly increased.

On the 5th of December, there was repeated vomiting, great debility, imperceptible pulse, and œdema of the lungs. Death on December 6th.

Autopsy, 25 *hours after death.*

The brain and cerebral membranes anæmic, but otherwise normal. In the right pleural sac were found about three pounds of clear serum; the surface of the left lung was everywhere firmly adherent. The mucous membrane of the air-passages was of a bright-red colour, and covered with small ecchymoses. The parenchyma of the lungs was bark-brown, œdematous, and somewhat firm in consistence; the lower lobe of the right lung was compressed; in the middle lobe was found a hæmorrhagic infarction the size of a walnut.

There were about seven ounces of serous fluid in the pericardium; the heart was considerably enlarged transversely; its right ventricle was dilated and hypertrophied, the hypertrophy being particularly marked in the *conus arteriosus;* the valves were normal; the left auricle was enlarged, and its muscular tissue thickened; the left auriculo-ventricular opening appeared narrowed to an oval slit, scarcely admitting the point of the forefinger; the margins of the valves, and a portion of the chordæ tendineæ adhered to a smooth thick ring. The muscular tissue of the left ventricle, and also the aorta, were normal.

There were about seven pounds of fluid in the abdominal cavity. The mucous membrane of the stomach, which was covered by a tenacious blackish substance, presented hæmorrhagic erosions, which were most numerous in the neighbourhood of the pylorus; its tissue was everywhere tumid and congested. The mucous membrane of the intestine, however, was throughout pale and relaxed.

The spleen was of normal size, dark-brown, and firm.

The liver was enlarged, its surface was slightly granular, and its margins sharp.* The gall-bladder contained a considerable quantity of thin bile, in which albumen was detected.

The left kidney was of normal size; small depressed scars were observed upon its surface, and its parenchyma was somewhat firmer than usual. The right kidney was much contracted; its surface was scarred and furrowed; its cortical substance had disappeared; a yellow infarctus the size of a sechser (or of a threepenny-piece) was observed upon its convex edge.

No important pathological change was found in the urinary bladder or genital organs.

In addition to the morbid conditions of the heart and lungs, which, from the fact, that the entire mass of the blood must pass through these organs, pre-eminently give rise to obstructions in this portion of the venous circulation, we observe, although with comparatively less frequency, mechanical congestions of the liver, produced by constrictions of the vena cava inferior above the opening of the hepatic veins, as also by constrictions of the hepatic veins themselves at their entrance into the vena cava. Watson† has described an instance of hyperæmic swelling of the liver, extending as low down as the crest of the ilium, which was caused by the lateral pressure upon the vena cava of an aortic aneurism. After the bursting of the aneurism, the pressure upon the vena cava ceased, the stagnating blood flowed on, and the organ had returned to nearly its normal size before the *post-mortem* examination was made. I have myself observed slight obstructions result from the compression of the vena cava by retro-peritoneal cancerous tumours. I have also seen a constriction of the hepatic veins caused by valvular projections into the interior of this vessel in a case of cirrhosis of the liver; in this case, the obstruction had given rise to numerous apoplectic patches in the parenchyma of the liver.

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* The changes in the parenchyma are represented in Plate XII., Figs. 1 and 4 of the Atlas.

† Principles and Pract. of Physic, 3rd Ed., Vol. I., p. 22.

However obvious the effects of the obstruction may be, it is seldom that we can arrive at a perfect knowledge of derangements of this nature during the life of the patient. This remark applies also to the partial mechanical hyperæmias, which are developed as a result of deep fissures in the liver from tight-lacing; and to the swellings of one lobe caused by induration of the other, or by obliteration of one of the branches of the portal vein, whereof I have before me several observations.

B. *Congestions and Atonic Hyperæmias of the Liver.*

We include under these designations the hyperæmias of the liver which are developed independently of any obstruction to the circulation of the blood. They are partly of an active character, and produced by exciting causes acting upon the parenchyma of the liver; but, on the other hand, they are partly of a passive nature, being occasioned by an atony of the vessels, or by a debilitated action of the heart,* and in this case, they are mostly of long duration. It is impossible, however, to draw a strict line of demarcation between these two forms, inasmuch, as we meet with undoubted examples of a transition from the one to the other, and because we cannot always ascertain with sufficient accuracy the mechanism of the affection in individual cases. It is equally impossible to determine to what extent the capillaries of the hepatic artery are implicated along with those of the portal vein; it can scarcely be doubted, that differences exist in this respect, when we consider the varying character of the influence which hyperæmias exercise over nutrition.†

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* The simplest forms of passive accumulation of blood in the liver, which I have observed, have been in old decrepit individuals with extreme prostration of the heart's action, and in whom the abdominal muscles, from being greatly relaxed, no longer possess the property of assisting the portal circulation. It was under such circumstances that Virchow correctly pointed out that the veins of the mesentery, spleen, and stomach, are distended with dark blood. (*Virchow's Archives*, Vol. V., p. 289.) The portal system is particularly liable to suffer from such derangements, owing to its double capillary circulation, and to the comparatively scanty muscular tissue and feeble contractility of its vascular walls.

† Hyperæmia of the liver from abuse of spirits soon leads to cirrhotic

Congestions of the liver constitute the starting-point of almost all the structural diseases of the organ. They also induce and accompany the formation of pseudo-plasmata. An accurate knowledge, and a careful attention to these conditions are particularly important in medical practice, inasmuch as at this stage of the disease such results may be hoped for from treatment, as at a later period it is usually vain to expect.

a. Congestions proceeding from the processes of Digestion.

Even in health, the quantity of blood contained in the liver is liable to constant fluctuations, arising from the process of digestion. The increased flow of blood to the mucous membrane of the stomach and the active intestinal absorption,—both of which circumstances are necessary accompaniments of this function,—increase the pressure of blood towards the liver, and give rise to a temporary swelling of the gland, which is compensated for by the augmentation of its secretion and by an increased consumption of plasma in the cells of its parenchyma. This hyperæmia not unfrequently exceeds the boundaries of health, as when acrid, irritating matters, such as alcohol, pepper, mustard, strong coffee, &c., are taken in too large quantity. The action of spirits in this way is the one of which we know most. These liquids, if taken very frequently, give rise at last to palpable changes of structure. In our climate, they gradually lead to cirrhotic degeneration; but in hot countries, according to the experience of Annesley,* Twining,† Cambay,‡ and others, they contribute greatly to the development of suppurative hepatitis.

The other irritating substances just mentioned, are less apt than spirits to be followed by injurious consequences, inasmuch as the ingestion of an excess of any of them is usually only a matter of occasional occurrence. Their action manifests itself chiefly by a feeling of tightness and fulness in the right hypochondrium, accompanied by a distinct enlargement of the gland, which can be made

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degeneration of the gland, whilst other forms of congestion may last for a long period without producing any such results. Moreover, the circulation in the hepatic artery often appears to become deranged, and black pigment is very frequently found deposited in the finer twigs of this vessel.

* ANNESLEY, *Diseases of India*. London, 1828. Vol. I., p. 488.

† TWINING, *Diseases of Bengal*, Vol. I., p. 247.

‡ CAMBAY, *De la Dyssenterie des pays chauds*, p. 217.

out by means of the pleximeter, and by pains which are wont to come on soon after meals, and which, as a general rule, cease after a short time. These results are chiefly observed during the warm seasons of the year, and in individuals who have already suffered from some affection of the liver, or in whom that organ is unusually irritable.

Beau* has, by a series of observations endeavoured to prove, that severe neuralgic pains in the liver can be produced by the same exciting causes as those just mentioned,—an attempt, which, in my opinion, can lead to no certain results, when we consider the difficulty of excluding the co-operation of gall-stones. We shall return hereafter to this matter in treating of Colica hepatis.

The question has frequently been discussed in what way acrid ingesta come to act upon the parenchyma of the liver. Broussais is of opinion that the irritation of the intestinal mucous membrane is transmitted along the bile ducts to the parenchyma of the liver; others have assumed the existence of an irritation sympathetic with that of the bowel; whilst Beau first of all pointed out the direct action of irritating substances by their absorption into the portal blood.† It is impossible to arrive at an accurate discrimination between these different modes of action; substances which, like alcohol, are readily taken up into the blood which is about to pass through the liver, produce their effects principally by way of absorption; other substances, again, which are less easily absorbed, transmit their effects from the mucous membrane along the nerves; a direct transmission of the irritation along the bile ducts, such as was assumed by Broussais, occurs much more rarely, and is attended in most cases by other consequences, involving the excretory ducts more than the glandular substance itself, as, for instance, catarrh of the bile ducts, obstruction of the secretion, &c.

The form of hyperæmia just alluded to, usually passes off without being productive of any great mischief; it is only from its recurring repeatedly, or from its co-existence with other irritating causes, that it leads to more serious disease; even then the affection is most frequently of a chronic nature.

This last condition of chronic congestion is particularly observed in

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[*] Archives général. de Méd. April, 1851.

[†] Afterwards, in treating of diseases of the portal vein, we shall consider a fourth modification of the mode of transmission of pathological processes from the intestine to the liver, namely, that through the veins, which was particularly pointed out by Ribes (*Andral, op. cit.*, p. 290).

individuals who devote themselves too freely to the pleasures of the table, and who make use of a succulent stimulating diet, notwithstanding their sedentary mode of life, their great muscular inactivity, and comparatively imperfect respiration. In such cases, the absorption of nutritive material exceeds the waste, and, sooner or later, but usually in the middle period of life, the muscles become flabby, and other affections show themselves in patients with a hereditary predisposition, while even at an earlier period there arises a disproportion between the power of the heart and the quantity of blood, owing to which that part of the vascular apparatus where the greatest obstacle has to be overcome, becomes over-distended with blood. This is usually the portal system, which is the more readily congested from the fact, that under the circumstances just mentioned the irritation of the intestinal mucous membrane, resulting from errors in diet, as also the copious intestinal absorption, contribute to the interruption of the circulation. In this way, one often sees chronic gastro-intestinal catarrh commencing with irregular, and, for the most part, retarded defæcation, and accompanied by swelling of the hæmorrhoidal veins, tympanites, and pains in the hypochondrium. To these symptoms there is frequently superadded a habitual hyperæmia of the liver, which from time to time increases and makes itself known through painful distention of the right hypochondrium, a jaundiced tinge of the conjunctiva, &c.* This condition may exist for a long time without inducing more serious lesions of this gland, such as fatty infiltration of the cells and catarrh of the bile ducts. In most cases, we may succeed without any difficulty in moderating or removing the hyperæmia of the liver by means of a carefully regulated diet, by avoiding all indigestible, fatty, and over-nutritious articles of diet, by increasing the waste of material by means of active exercise in the open air, riding, &c., as well as by the use of bitter laxative medicines, the solvent extracts with salines, rhubarb, aloes, and similar substances, by the application of leeches to the anus, and still better, by regulating the intestinal secretions by the use of the Springs of Kissingen, Homburg, Marienbad, and Karlsbad †; but if the condition is one of very old-standing, it is seldom that the improvement is permanent. It would appear that the nutrition of the muscular tissue of the intestinal canal, and probably also of the lining membrane of the portal veins becomes at an early period impaired, and as a result of this, there is induced a predisposition to relapses, and the patient's complaints are protracted. Accurate examinations of the anatomical characters of the tissues just mentioned are necessary, in order to confirm and explain those circumstances which hitherto have been made the subject of theoretical speculation. Along with the mechanical derangements which have been pointed out, we usually find abnormal conditions of the metamorphosis of matter, which were formerly attributed to arthritis, to the formation of urinary gravel, &c. Our knowledge of these conditions deserves to be placed upon a firmer footing than it is at present; we shall afterwards see how far the tissue of the liver may be implicated in their devolopment.

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* Every extensive development of gas in the intestinal tube, by pressing the blood from the roots of the portal vein towards the trunk of that vessel and the liver, may induce transient hyperæmia of this organ; great accumulations of fæcal matter may give rise to similar results.

† See page 125, note.—TRANSL.

This is not the place to discuss in detail the various morbid processes which medical men have included under the name of abdominal plethora; when the liver is implicated, which, however, is by no means the general rule, there is in most cases nothing more than hyperæmia, or fatty degeneration (*see Chapter on Fatty Degeneration*, page 306); more serious lesions, such as cirrhosis, induration, or lardaceous degeneration are rarely present. The mutual relations which exist between these affections and the condition of the intestinal mucous membrane, will be understood from what has been already stated.

b. Traumatic Hyperæmia.

Contusion of the hepatic region is not unfrequently observed to give rise to accumulations of blood in the liver, entailing a considerable amount of swelling of the organ. Piorry has described a case of this nature resulting from the blow of a spent pistol-ball; the remarkable increase in the volume of the gland, which was accompanied by dyspnœa and fever, disappeared after a copious venesection, so soon as the following day. I have myself met with a similar enlargement of the liver in a railway-labourer, the right side of whose chest had been crushed by a waggon. The recovery in this instance was more tedious; the patient continued jaundiced for three weeks, at the end of which time he was dismissed from the Hospital cured. In many cases, the effects of the contusion are restricted to the part; the hyperæmia may then pass readily into inflammation, which may lead insensibly to the formation of abscess. Most of the hepatic abscesses which occur in our own climate, have this traumatic origin. R. Bright has published some cases, which appear to prove that the hyperæmia resulting from external violence may pass into a chronic form of inflammation, ending in induration.

c. Hyperæmias dependent upon the influence of a high temperature and miasmatic effluvia.

In hot climates, and particularly in those marshy districts which exhale deleterious substances, hyperæmias of the liver, and the injurious consequences resulting therefrom, are among the ordinary forms of disease. In addition to the high temperature, the miasmatic infection of the blood contributes very powerfully to their development; but it is a difficult matter to determine the share which is taken by each of these two agents. There are facts which would seem to show that the influence of heat has been overestimated, owing to the idea, that in a high temperature the liver acts vicariously with the lungs. Haspel states, that in the year 1846, when the temperature in Oran was unusually high, so that the marshes were dried up and the sources of malaria destroyed, hepatic affections, in place of being more prevalent, completely ceased. In India, the prevalence of hepatic diseases depends less upon the temperature of the stations, than upon their situation in the neighbourhood of stagnant water and marshes. Hepatic affections usually attain their greatest frequency in autumn, when the temperature is beginning to fall. In the cold foggy climate of Holland, Pringle found conditions of the liver precisely similar to those met with in the tropics. Malaria must, therefore, be regarded as the principal exciting cause of hyperæmia of the liver, although the co-operation of a high temperature cannot be doubted.

The hyperæmia of the liver, which is wont to be developed in warm climates, usually attains a remarkable intensity, and, not unfrequently, in a short time induces serious structural changes. The gland is distended with dark blood; the gall ducts and bladder are filled with secretion, and in this condition hæmorrhages and sub-peritoneal effusions take place in the liver, which may end in softening of the parenchyma; in other cases, limited inflammatory deposits are formed, which, after a shorter or longer period, terminate in abscess; or derangements of nutrition arise, which give rise to hypertrophy or fatty degeneration, or, on the other hand, to induration, or, less frequently, to cirrhotic degeneration with shrinking of the parenchyma.*

The hepatic hyperæmias of the tropics are often uncomplicated, but more frequently they make their appearance along with dysentery, or in conjunction with malarious fevers of an intermittent, remittent, or continuous type. In the latter case, hyperæmic swellings of the spleen, and often of the kidneys also, co-exist with the hepatic affection, and in this way the various complications originate, the pathology and symptoms of which we have already briefly alluded to.

Simple hyperæmia is either acute or chronic in its progress; the derangements to which it gives rise are more marked in the former case than in the latter. Chronic hyperæmia is usually developed so slowly and insidiously that its existence is frequently not recognised until serious structural changes in the hepatic tissue, such as abscesses, induration, &c., take place, and remove all hopes of a favourable result from treatment.

The acute form of hyperæmia is characterised by more or less painful distention of the right hypochondrium, accompanied by dyspnœa, and often, also, by tension of the splenic region, and pains stretching towards the right shoulder and lumbar region. Along with these symptoms, the tongue is either clean or sometimes coated grey, and there is headache, nausea, and vomiting of mucous or green bilious matter: the bowels are irregular; in some cases they are constipated, but more frequently there is diarrhœa, with yellow, bilious, or sometimes even bloody stools: at the same time, there is great depression of spirits and rapid loss of strength, in most cases, without any increased frequency of pulse or elevation of temperature. After some days or weeks the symptoms quite disappear, or slight derangements remain, obvious only to an attentive observer, and denoting the transition of the acute into the chronic form.

Although the unpleasant sensations in the right hypochondrium may abate or disappear, the hepatic dulness still remains extensive; the appetite only partially returns, some articles of diet, particularly animal food, excite loathing, and the tendency to diarrhœa continues. These symptoms are subject to exacerbations from time to time; the hepatic tumour increases, and the gastric and intestinal derangements become more marked. Amidst alternations in the severity of these symptoms, the patients emaciate more and more, and become sad and desponding; their skin assumes a pale cachectic appearance, or sometimes there is jaundice; and dropsical effusions are poured out into the abdominal cavity, and into the subcutaneous areolar tissue. Towards the termination of the disease, there are frequently developed exhausting catarrh of the intestines, dysenteric affections, and intermittents of an irregular type, accompanied by suppuration of the parotid, &c.; or symptoms of suppurative fever, indicating the formation of abscess in the liver, may show themselves.

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* The deposits of pigment and their consequences, which one meets with under such circumstances, have already been treated of in Chap. VIII.

These symptoms of the final stage of chronic hyperæmia are mainly dependent upon the nature of the structural changes which the organ undergoes during the progress of the affection, and vary in accordance with the varying character of these changes. Upon *post-mortem* examination, the liver is sometimes found to be congested and softened, at other times it is pale and jaundiced or fatty, or it may be indurated and cirrhotic, or lastly, it may be penetrated by abscesses. The study of the clinical history of these pathological changes will explain the varieties in the symptoms induced by chronic hyperæmia of the liver.

In temperate climates, the affection usually runs a milder course, the symptoms are less prominently marked, and the modes of termination are different. Hyperæmia of the liver may exist here for a long period, before it gives rise to derangements of nutrition of any consequence; abscesses are scarcely ever met with. What we most frequently observe is an increase in the volume of the liver, caused by fatty deposit, or by an infiltration of the parenchyma with an albuminous substance, which gradually passes into colloid degeneration; in rare cases the liver becomes cirrhosed. Acute hyperæmia of the liver is especially observed during the hot summer months; chronic hyperæmia, with its sequelæ, is not usually developed until autumn.

The first object, in the treatment of the acute form, is to remove everything which can act by stimulating the action of the liver. The diet is to be restricted to mild demulcent or somewhat acid vegetable substances; animal food, fat, strong spices, and alcoholic drinks are to be avoided. The reduction of the excess of blood in the portal system and in the liver, is best effected by the application of leeches to the anus, tepid baths, and cooling saline purgatives, such as 'amarind pulp, purified tartar, and sulphate of soda. If spontaneous diarrhœa sets in, it must not be checked too early; when it is necessary to interfere, it is best to make use of ipecacuanha, either in small divided doses, or as an emetic. The general compression to which the liver is subjected during the act of vomiting, has a great effect upon the circulation of blood through the organ. The mineral acids may also be prescribed.

When the affection is of a chronic or passive character, change of air is advisable, taking care to avoid all marshy districts. In such cases, in order to promote intestinal absorption, small doses of rhubarb, aloes, colocynth, &c., in combination with the sesqui-chloride of iron, and the bitter solvent extracts (page 123), will be found most serviceable. An emetic may also be tried here with the object of assisting, mechanically, the circulation of the blood in the hepatic capillaries. A too strict diet is not adapted to this form of the affection: easily digested nutritious food is necessary in order to improve the condition of the blood and correct the atony of the vascular apparatus; in more advanced cases, preparations of steel are usually employed for the same purpose. Where circumstances permit, a resort to Kissingen, Homburg, or Marienbad* is advisable. Pringle, Lind, Portal, and Haspel have advised the application to the hepatic region of blisters, setons, and moxas, and that they should be kept open for a long period. Calomel and mercurial ointment have been frequently recommended, but must be resorted to with caution when a cachectic condition threatens to become developed. In cases where the hepatic affection is accompanied by irregular forms of intermittent fever, quinine must not be employed too hastily; this febrifuge is usually of little service until the hyperæmia of the liver has been subdued by other means.† When there is reason to apprehend from the symptoms, that an abscess is forming, or that lardaceous degeneration, or cirrhosis, is being developed, the treatment appropriate to each of these conditions must be adopted.

Closely allied to the chronic hepatic hyperæmia of malarious regions, is the congestion of the liver which is observed in scurvy, and in similar conditions of the system, along with swelling of the spleen. Baillou‡ and Portal§ long ago described cases of extreme distention of the hepatic vessels with dark blood, occurring in scorbutic subjects, and giving rise to softening of the glandular tissue. Andral* never failed to find this condition under such circumstances. According to my own observation, this morbid change in the dead bodies of scorbutic patients has not been of such constant occurrence; I have repeatedly found the organ soft, infiltrated with fat, and anæmic. Portal has recorded cases in which he obtained favourable therapeutic results from the employment of anti-scorbutic remedies combined with mild laxatives.

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* See note, page 125.—TRANSL.

† See ANDRAL, *op. cit.*, p. 31; and also PORTAL.

‡ BALLONII, *Opera*, Tom. III., p. 30.

§ PORTAL, *op. cit.*, p. 377.

d. Hyperæmia of the Liver, from the Suppression of habitual Hæmorrhages.

During the climacteric period of life, the cessation of the catamenia is not unfrequently observed to be accompanied by tumefaction of the liver, which disappears with each recurrence, after a long interval, of the uterine hæmorrhage; and in this way, the swelling may return repeatedly. Like consequences are often noticed in the earlier periods of life from sudden suppression of the menses; Portal, and more recently, Henoch,† have published cases of this nature. The cessation of hæmorrhoidal discharges of blood, which have become habitual, may be followed by similar results. It has been ascertained, however, that this effect is by no means constant; in many cases, the liver remains quite unaffected; hence, other causes must co-operate in producing these congestions. So far as my knowledge extends, this form of hyperæmia of the liver has not been observed to give rise to any serious derangement in the nutrition of the liver; it is obvious, however, that pre-existing diseases of the liver may be aggravated and hastened in their progress by it, and hence, in treatment, care should be taken to reproduce at an early period the original hæmorrhages, or to obtain some compensation for them.

There can be no question that there are other causes of hyperæmia of the liver, besides those already mentioned, with which we are either imperfectly, or not at all, acquainted, and that, therefore, it must very frequently happen that the cause of such an affection must remain doubtful. Hence, the forms of hyperæmia hitherto considered are not all, but merely the most important. There are three agencies in particular, which may serve for the further elucidation of these affections. First, there is the influence of the nerves, which has been in some measure experimentally proved by Claude Bernard and others, although it has not been studied by any means sufficiently, in order to be made use of in clinical investigations. That this influence may operate powerfully under certain circumstances, is apparent from the cases of diffuse inflammation and acute atrophy of the liver, which are developed immediately after mental emotions.* A second agency, although one that is but imperfectly understood, is the composition of the blood, the importance of which, in malarious infection, in scurvy, &c., has already been pointed out, but which probably assists in many other cases, in producing hepatic hyperæmia. Thirdly and lastly, the altered nutrition of the walls of the hepatic vessels, which hitherto has attracted but little attention, promises to explain many points, not only in the chronic forms of hyperæmia of the liver, which are for the most part dependent upon atony of the blood-vessels, but also in the partial congestions and apoplexies of the organ. These causes of hyperæmia must be reserved for further investigation.

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* Andral, *Clinique Médicale*, T. II., p. 244.

† Henoch, *op. cit.*, p. 85.

As an appendix to the hyperæmias, we shall consider as briefly as possible:—

Hæmorrhage from the Liver.—Apoplexy and Hæmorrhagic Softening of the Liver.

These affections are in general of rare occurrence, and are for the most part the result of intense congestions, which are induced by miasmatic infection of the blood. Hence, they are particularly observed as a complication of the malarious fevers of the tropics, and occasionally, also, of the malignant intermittent fevers of colder climates. The extravasated blood is sometimes accumulated in isolated masses, or circumscribed beneath the serous covering of the gland, whereas, at other times, the parenchyma is more uniformly infiltrated with blood, and converted into a dark, abnormally soft, and at some places, pultaceous mass, in which the remains of the normal tissue can only be sparingly recognised. The gall-bladder usually contains a dark, thick secretion.† This condition is accompanied during life by the symptoms of malignant marsh fever, by pains in the right hypochondrium, and almost always by jaundice and bilious vomiting; the co-existence of hæmorrhages from other parts of the body is not uncommon. Annesley, Haspel, and others record many examples of this nature.

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* See above, page 234.

† It has not yet been determined with sufficient accuracy to what extent the extravasated blood enters the hepatic ducts and is conveyed by

Similar morbid changes in the liver have occasionally been observed in scurvy and allied affections. Extravasations of blood in the hepatic tissue, or beneath its capsule, are also, frequently caused by external violence, and occasionally by obstructive hyperæmia; they are particularly observed in new-born children after tedious deliveries, and in conjunction with atalectasis of the lungs.*

There are some very remarkable cases of hæmorrhage of the liver, where, without any pre-existing complaints, the bleedings have come on suddenly, and in a short space of time have terminated in death. Andral† records instances of this nature, which do not admit of any certain explanation. One case was that of the Superintendent of the Mint at Paris, who, on awaking in the morning, complained of slight uneasiness, and expressed a desire to remain in bed. Some hours after, he was found dead. On *post-mortem* examination, the organs of the head and chest appeared perfectly normal; the abdominal cavity contained a large quantity of partially coagulated blood. In the middle of the convex surface of the right lobe of the liver, an opening was visible, the size of the point of the finger; this led into a cavity, the size of a hen's-egg, filled with blood, at the bottom of which was detected the ruptured wall of a large branch of the portal vein; in other respects, the liver was healthy. Andral unfortunately makes no mention of the condition of the coats of the vena porta. In another case, described by Honoré, the liver contained several cavities filled with blood; no search for a ruptured vessel was made. Louis‡ found in the liver a cavity the size of a nut, filled with

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them to the gall-bladder and intestine, or whether it does so at all. Saunders believed that he had found blood in the bile in several cases; this secretion was almost black, coagulated, and deposited, upon the addition of water, a sediment consisting of red corpuscles.

* In a stillborn child, with hernia umbilicalis congenita, I have found upon the convex surface of the left lobe of the liver a sub-peritoneal extravasation of blood, 1¼ inch in diameter, and at the same time smaller extravasations in the substance of the gland. See further, F. Weber, *Beiträge zur patholog. Anat. der Neugeborenen*, Bd. III., S. 56.

† ANDRAL, Clinique Médicale, T. II., p. 247.

‡ LOUIS, Recherch. anatomico-patholog., 1826, p. 381.

coagula of blood arranged in concentric laminæ. It is probable, that these hæmorrhages depend upon local diseases of the vascular coats, which have not yet been sufficiently examined. In the Second Volume of this work, I shall communicate a case of fatty degeneration of the coats of the portal vein, which occasioned rupture of this vessel before its entrance into the liver, and, as a consequence of this, fatal hæmorrhage. The coats of this vein may be subject to similar derangements of nutrition in the interior of the liver.

One additional case of hæmorrhage from the liver may be here mentioned in detail.

OBSERVATION No. XLVIII.

Tedious Delivery.—Symptoms of Peritonitis.—Jaundice.—Vomiting of black flakes.—Delirium.—Death.

Purulent effusion in the peritoneal cavity.—Hæmorrhagic softening of the Liver.—Extravasation of blood beneath the capsule.

Caroline Herbst, aged 38, on January 10th, 1856, was confined for the twelfth time; labour was tedious. The very day after, vomiting and diarrhœa, with suppression of the lochial discharge, made their appearance.

On the patient's admission into Hospital on the 18th, the abdomen was found remarkably distended and tympanitic; dulness upon percussion could be made out in the iliac regions; there was a slight amount of pain; the uterus extended a hand's-breadth above the symphysis of the pubes; the liver and spleen were of normal dimensions; the stools were brown, hard, and scybalous. The organs of the chest were normal; pulse 110 and small. Was ordered tamarinds with sulphate of soda and warm cataplasms.

On the 19th, the bloody discharge from the genital organs returned; meanwhile, the right hypochondrium had become painful; the pulse had risen to 120, and there was slight diminution of the tympanites; the conjunctiva and the countenance had assumed a jaundiced hue. The bowels had not been moved. Continuation of the same remedies, and an enema.

On the 20th, the jaundice was very intense, and was accompanied by typhoid somnolence, great prostration, and repeated vomiting of brown flaky masses. The urine drawn off by catheter abounded in bile-pigment, was acid, and free from albumen, and had a specific gravity of 1014. Was ordered phosphoric acid and analeptics. The tympanites and somnolence increased, and death ensued on the following morning.

Autopsy, 17 *hours after death.*

The body was of a dark-yellow colour; there were a few patches of putrefaction. There were no important morbid changes in the organs of the head or thorax; the heart contained dark, firmly coagulated blood. A considerable quantity of purulent fluid distinctly tinged with bile was effused into the peritoneal sac; the peritoneum was opaque and injected.

A greyish-yellow fluid, mingled with black flakes, was found in the stomach; the mucous membrane was pale, and free from ulceration; the intestinal canal contained at its upper part, thin and yellow, and at its lower, firm and brown, fæcal matter; the mucous membrane was normal.

The spleen was unaltered in size and consistence; it weighed 0.17 kilogr. (6 oz. avoird.). The kidneys were of a jaundiced tint, but in other respects normal.

The uterus extended about three inches above the symphysis pubis, and its inner surface was covered with a reddish-brown fluid, but presented no morbid change of any consequence; the veins and lymphatics were empty; the left ovary contained a cyst the size of a walnut, filled with a clear fluid; the vagina was of a livid hue, without any exudation.

The liver appeared enlarged, it weighed 2.3 kilogr. (5 lbs 1⅛ oz. avoird.); its form was not much altered; its consistence was soft and flabby, and some parts of the right lobe and lobulus quadratus felt pultaceous. Beneath the serous covering, numerous extravasations of blood were observed, varying in size from that of a groschen (6¼ Eng. lines, or rather less than a sixpence), to that of a thaler (1¼ inch, somewhat larger than a half-crown); a flabby, bladder-like elevation of the capsule, filled with bloody fluid, and more than an inch and a-half in diameter, was situated at the outer margin of the right lobe. Numerous large and small extravasations of blood were found in the parenchyma of the gland; the glandular tissue was at some places reddish-yellow or yellow, and exhibited listinctly the outlines of the lobules; at other places, it was of a ty reddish-brown colour. A large quantity of bloody serous fluid exuded from the cut surface, especially from the deeply injected portal margins of the lobules. The hepatic cells in the centre of the lobules were normal; at the periphery they were mingled with a quantity of debris and free nuclei; many of them contained oil globules; there was very little bile-pigment, either in a granular, or in a diffused form.

The bile ducts contained a little pale-yellow mucus; and the gall-bladder, a small quantity of thick, greenish-yellow bile, free from albumen. The branches of the portal vein, which were traced as far as was practicable, were of normal character, and the fluid blood contained in them presented nothing unusual under the microscope.

This case, so far as regards the lesion of the hepatic tissue, was allied to the changes which the liver is wont to undergo in the fevers of tropical climates and in scurvy. No cause, however, of this nature could be made out, while the idea of a putrid infection of the blood was countenanced by neither the symptoms nor the anatomical lesions. The intense hyperæmia of the liver could not be attributed to the suppression of the lochia, because the jaundice did not make its appearance until this discharge had returned. It would seem that the same condition was present in this case, as, in a milder form, not unfrequently induces diffuse hepatitis and acute atrophy of the liver in pregnant females. The incipient disintegration of the hepatic cells at the margin of the lobules, the deposit of fat in these cells, and the infiltration with fluid exudation, are circumstances which render this view of the case more probable than any other.

APPENDIX

OF OBSERVATIONS AND EXPERIMENTS IN SUPPORT OF THE STATEMENTS MADE IN THIS WORK.

I. *Observations of Disease.*

No. I. (*Page* 55.) Tertiary syphilis—Tumour of the liver, which ascended as high as the second rib, and consisted of echinococci, together with lardaceous infiltration—Bulging and fluctuation of the intercostal spaces—Displacement of the heart—Immobility of the tumour upon deep inspiration—Diagnostic value of exploration by a trocar.

No. II. (*Page* 65.) Cancer of the small omentum—Compression of the portal vein and atrophy of the liver—Ecchymoses in the serous coat of the intestines and in the parietal peritoneum.

No. III. (*Page* 73.) Cancer of the right kidney—Displacement of the liver upwards and to the left.

No. IV. (*Page* 104.) Pneumonia duplex—Icterus—Bilious stools—Green expectoration, continuing for ten days after the cessation of the pneumonia, and for eight days after the disappearance of the jaundice from the skin.

No. V. (*Page* 132.) Dyspepsia—Symptoms of chronic simple ulcer of the stomach—Jaundice—Distention of the gall-bladder—Pleurisy on the right side—Dropsy—Petechiæ—Death.

Cancer of the duodenum and dilatation of the bile ducts—Simple ulcer of the stomach—Exudation in the right pleural cavity.

No. VI. (*Page* 137.) Cancerous deposit in the head of the pancreas and in the duodenum—Occlusion and widening of the bile ducts and of the pancreatic duct—Distention of the bile ducts, with mucus and bile—Dysentery—Diminished secretion of urine—Infiltration of the kidneys with solid deposits of bile-pigment—Death from exhaustion.

No. VII. (*Page* 145.) Cancerous deposit in the head of the pancreas —Occlusion of the ductus choledochus and of the pancreatic duct—Enlargement of the pancreatic duct and of the bile ducts—Jaundice—Intestinal hæmorrhage—Diabetes mellitus —Dysentery—Death from exhaustion.

No. VIII. (*Page* 151.) Closure of the ductus choledochus by newly-formed areolar tissue, the result of Peri-hepatitis—Jaundice and enlargement of the bile ducts—Dropsy—Secondary pneumonia—Death.

No. IX. (*Page* 162.) Contusion of the pelvic bones—Rigors—Somnolence—Jaundice—Albuminuria—Death.

Phlebitis of the pelvic veins—Metastatic deposits in the lungs—Soft anæmic liver.

No. X. (*Page* 164.) Acute articular rheumatism—Endocarditis—Repeated rigors—Painful enlargement of the spleen—Jaundice —Albuminuria and hæmaturia—Petechiæ—Convulsions—Coma, and death.

Recent deposits upon the mitral valve—Splenic infarctions—Flabby anæmic liver—Ecchymoses upon the mucous membrane of the intestines, bronchi, &c.

No. XI. (*Page* 168.) Exanthematic typhus—Jaundice—Albuminuria—Hæmorrhage from the bowels—Ecchymoses of the skin —Parotitis—Death on the twelfth day.

Small spleen—Anæmic liver—Normal hepatic cells—Unobstructed bile ducts—No disease of the intestine, nor of the mesenteric glands.

No. XII. (*Page* 170.) Petechial typhus—Jaundice—Albuminuria Suppression of urine—Right pneumonia—Dysentery—Death on the seventh day.

Lardaceous spleen of old date—Anæmia of the liver—Exudation into the right lung—Dysentery—Recent exudation in the kidneys.

No. XIII. (*Page* 172.) Abdominal typhus—a severe rigor during convalescence—Fresh enlargement of the spleen—Great tenderness in the region of the liver, and afterwards of the entire abdomen—Jaundice—Dyspnœa—Somnolence—Death.

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No. XLVIII. (*Page* 385.) Tedious delivery—Symptoms of peritonitis—Jaundice—Vomiting of black flakes—Delirium—Death. Purulent effusion in the peritoneal cavity—Hæmorrhagic softening of the liver, and extravasation of blood beneath the capsule.

II. EXPERIMENTS IN SUPPORT OF THE THEORY OF ICTERUS, ADVOCATED IN THIS WORK.

Injection of pure bile into the blood of living animals.

In order to ascertain the effects upon the various functions resulting from the absorption of a large quantity of bile into the blood, but particularly with the object of tracing what becomes of the bile in the blood,—whether it is transformed, or is excreted as bile, and, if transformed, what may be the nature of the changes it undergoes,—a series of experiments were undertaken, the results of which are here given. Fresh ox-bile,—from which the mucus had been removed by means of alcohol, and the whole of the colouring-matter, by animal charcoal,—was employed for injection. Two or three, and in some cases, four or five, grammes* of dried bile were dissolved in from thirty to forty-five grammes of distilled water, and filtered; the colourless or pale-yellow fluid was then carefully injected into the jugular veins, or more rarely, into the veins of the thigh, a quantity of blood corresponding to that of the injected fluid, having previously been drawn off by the opening in the vein, so as to prevent the vascular system being over-distended. In some cases, the inspissated bile of the Prussian Pharmacopœia was employed in place of fresh bile. Dogs were the animals on which the experiments were always performed; in most instances, they had been previously etherised to a slight degree; but by the time of the injection the action of the ether had ceased, and could no longer mask any derangements of the nervous system, which might make their appearance. After the injection, the animals were placed in a box constructed for the collection of the urine, and carefully watched. In most cases, however, the urine was poured directly into the utensil by compressing the bladder, so that the possibility of the admixture of any foreign matter was completely excluded. Some of the animals died under symptoms of violent dyspnœa (obstruction of the capillaries of the lungs), owing to the entrance of air into the lungs or to the too great consistence of the injected fluid; in other cases the urine passed into the box, was soiled; twenty-nine of the experiments succeeded in such a way that their results could be made use of.*

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* 1 gramme = 15.4325 troy grains.

1. *Effects of Bile introduced into the Blood upon the functions of the Nervous System.*

In no case did any remarkable derangements of the nervous functions follow the injection. As soon as the bile entered the blood, the animals usually appeared, by licking with the tongue, to experience a change of taste; but in no case were stupor, convulsions, retardation of the pulse, &c., observable; vomiting alone occurred repeatedly (perhaps in one-fourth of the experiments), and particularly in those cases where the inspissated ox-bile was employed; in some of these cases, also, the experiment was followed by some drowsiness. A short time after the injection, so little that was unusual could be observed in the behaviour of the animals, that in one the operation was performed four times in different veins, without being productive of any lasting injury.

2. *Changes in the urine.—Appearance of bile-pigment, &c.*

The character of the urine which was voided after the injection varied; sometimes it contained a larger or smaller quantity of colouring-matter, and at other times it contained none; the former was the case in 19, and the latter in 10, of the 29 experiments.

The urine containing colouring-matter was always passed in small quantity; it was of a greenish-brown colour, became turbid upon cooling, and then appeared green, rapidly depositing flakes, which, under the microscope, presented a finely granular appearance. When collected upon a filter, this deposit formed a dark, grass-green layer, which dried readily, and which, when recently dried, exhibited the characteristic properties of the colouring-matter of bile, becoming decomposed by the action of impure nitric acid, or by a mixture of sulphuric and nitric acids, with a lively play of colours of green, blue, violet, and red. The flakes which were suspended in the urine presented unmistakably the same play of colours, although in a fainter degree. The reaction of the urine was, in most cases, neutral or alkaline, the removal of the flakes of colouring-matter was speedily followed by an abundant deposit of triple phosphates; the specific gravity of the urine varied from 1012 to 1019. In 17 of the 19 cases the urine was albuminous, and after the separation by filtration of the green flakes, its colour was blood-red, apparently owing to dissolved blood-pigment; no blood corpuscles could be detected in the sediment. In two cases mere traces only of albumen could be found, along with an abundance of colouring-matter. This altered urine was voided from four to twenty hours after the injection; in one case, in which there was obstinate retention of urine for forty-eight hours, the colouring-matter was very distinct; but in most cases the urine, excreted from twenty-four to forty-eight hours after the injection, had regained its normal characters.

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* I omit here the experiments which I performed seven years ago at Göttingen and Kiel with filtered ox-gall; inasmuch as the changes in the urinary secretion were not investigated, and, consequently, they are of but limited value.

3. *Chemical examination of the urine, passed within 24 hours of the injection of bile.*

a. *Examination of the urine which contained colouring-matter.*

In five of the nineteen cases, the urine was freed by filtration from the pigment flakes, evaporated to dryness, and the residue extracted by means of ether, and afterwards by alcohol. In no case, did the ethereal extract contain an amount of colouring-matter deserving of mention, but only traces of a yellow fat. The alcoholic extract was coloured brown, and did not usually exhibit the reaction of bile-pigment; in one case only, where the animal had died of dyspnœa some hours after the injection, did the greenish-brown colouring-matter obtained by means of alcohol, and which upon evaporation separated in the form of flakes, present the properties of bile-pigment. This alcoholic extract, when reduced to the consistence of syrup, exhibited the coloured rings characteristic of the chromogenic ingredients of bile; and green, blue, violet, and red rings were observed to be formed upon the vessel by the action of atmospheric air during evaporation.

Pettenkofer's test with sugar and sulphuric acid failed to detect any of the biliary acids.

In four of the five cases, the alcoholic extract contained leucine in considerable quantity.

In three cases crystals were obtained, which, from their characters, must be assumed to have been taurine, but their quantity was too small to admit of their being further examined.

In five cases the urine filtered from the flakes was treated with neutral and basic acetate of lead; the precipitate was dried, digested with alcohol and boiled. The alcoholic solution, which only took up small quantities of the compound of lead, was freed from lead by means of solution of sulphuretted hydrogen and concentrated. There remained a substance like myeline, and likewise leucine in the form of globules, composed of concentric layers, or of drusic masses made up of acicular crystals, together with isolated crystals of urea. The urea as well as the leucine appeared to be left, owing to the imperfect washing-out of the lead-compound. The mass was repeatedly re-crystallized, but no crystalline forms, corresponding to those of the acids of the bile, ever made their appearance, nor did Pettenkofer's test in any case yield a distinctly positive result.

In order to prove the certainty with which the biliary acids could be detected in the urine by treatment with lead, a small quantity of ox-bile freed from colouring-matter, was dissolved in healthy urine; the greater portion of it could be obtained again by precipitation with lead; and the alcoholic extract of this precipitate, when freed from lead, presented distinctly the characteristic reaction with Pettenkofer's test. We may therefore conclude, that in the above experiments the biliary acids were not excreted in an unchanged condition in the urine.

b. Examination of the urine, which was passed free from colouring-matter after the injection of bile into the blood.

Of the ten cases in which the urine was passed clear and pale, there was invariably a remarkable increase of the secretion; the quantity voided in twenty-four hours not unfrequently amounted to about 1000 cubic centimètres (35 fluid ounces). This urine was carefully examined in eight cases. The specific gravity varied from 1008 to 1012: on the addition of nitric acid, the pale-yellow colour was not changed in any way, so as to remind one of bile-pigment; no albumen was precipitated. In order to isolate any biliary acids which might have passed over unchanged, the secretion voided within the first twenty-four hours was precipitated by neutral and basic acetate of lead; the precipitate was collected and dried, and digested and boiled in alcohol; and then the filtrate was freed from lead by means of solution of sulphuretted hydrogen, and condensed. The quantity of the compound of lead, soluble in alcohol, appeared to vary according to the longer or shorter time that the precipitate had been washed with distilled water; in the former case, the quantity was small, in the latter it was large. The residuum remaining after the removal of the lead, and the evaporation of the spirit of wine, exhibited in five cases very beautiful crystals of leucine, and twice, where the washing had been only superficial, urea also; the so-called myeline, was usually seen to accompany these substances. When treated with sugar and sulphuric acid, in two cases only did the residuum yield the red colour indicative of traces of the acid of bile; in the other cases, there was a negative result. In one instance the colourless spirituous solution, after being freed from lead, exhibited, shortly before complete desiccation over the water-bath, the colour of the chromogen of bile; in this instance, too large a quantity of free sulphuric acid was not present; at least leucine was found undecomposed in the residuum.

In one other case, where the urine, which was free from both colouring-matter and albumen, was at once condensed, flakes of a green colouring-matter were formed during evaporation, which with nitric acid presented the same reaction as was observed in the spontaneous precipitate of the first nineteen cases. This appearance was not produced in two experiments which were subsequently made in the same way.

In many of the cases the urine was precipitated by lead, and after the removal of the excess of this metal, evaporated. Isolated globules of leucine could usually be found in the residue, which was loaded with urea, but sometimes they were searched for in vain in the mass of other crystals.

The following propositions result from the preceding series of experiments:—

1. The injection of large quantities of bile into the blood of living animals is followed by no important derangement in the vital functions.

2. After the injection into the blood-vessels of large quantities of bile deprived of its colouring-matter, pigment is in most cases excreted along with the urine, which exhibits most of the

characteristic properties of bile-pigment.* This colouring-matter closely corresponds in its behaviour with the products, which may be formed artificially by the action of sulphuric acid upon the acids of the bile.

3. In rare cases, in place of the colouring-matter, we find chromogen substances, which are not transformed into colouring-matter until the evaporation of the urine upon exposure to the air.

4. Unchanged biliary acids are not found along with the colouring-matter, but leucine is usually present.† Taurine and glycine cannot be detected with certainty.

5. The injection of bile is, in some cases, not followed either by the excretion of colouring-matter with the urine, or by the appearance of large quantities of unchanged bile.

The causes of this anomaly are unknown; the failure in the appearance of the pigment is accompanied by a profuse secretion of urine, whilst its presence is almost always associated with albuminuria, and often also with hæmaturia. The appearance of the colouring-matter appears to be favoured by whatever interferes with the respiration, at all events, it was particularly abundant in one animal, which died asphyxiated some hours after the injection, as also in two other animals, which had oil introduced into their air-passages with the object of obstructing their breathing.

The theory of that form of jaundice, which occurs without any structural lesion of the liver or bile ducts, which has been already discussed (page 89), mainly rests upon these observations. It is obvious, that defects still remain to be filled up, before this view can be regarded as firmly established. The only point proved with certainty, is the possibility of the transformation of the biliary acids into colouring-matter, and the dependence of this transformation upon certain conditions, which appear to consist principally in the functional activity of the respiratory organs and kidneys. When these conditions are more accurately determined, the individual causes of the metamorphosis of bile in the blood, and the connection of the various pigments with one another, must be better understood than hitherto has been the case.

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* The insolubility of the pigment constitutes an apparent difference. The colouring-matter, however, of the urine of jaundiced patients frequently exhibits the same character; in many cases, it can be completely separated from the fluid by filtration.

† Experiments, with the object of detecting leucine in decomposing bile, have failed for the most part in summer; but a large quantity of leucine, along with crystals of taurine, have been obtained from human bile which has been for a week in a freezing state during winter. The quantity of leucine which is obtained through precipitation of the biliary acids by the neutral and basic acetate of lead, appears much too considerable to be probably induced by the decomposition of the mucus of the bladder.

III. *Experiments on the rapidity of the absorption of bile after ligature of the ductus choledochus.* See page 94.

IV. *Experiments on the influence of a fatty diet on the generation of fatty liver.* See page 281.

V. *Experiments on the injection of oil into the portal vein.* See page 282, note.

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